

FRACTURES
AND
JOINT INJURIES

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FRACTURES AND JOINT INJURIES

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Hugh Owen Thomas
1834-1891

Sir Robert Jones
1837-1933

They, whose work cannot die, whose influence lives
after them, whose disciples perpetuate and multiply
their gifts to humanity, are truly immortal

PREFACE

The first edition of *Fractures and Joint Injuries* " was written under the threat of war, the second with the realisation of war, and the third after the experience of war. The writing of many pages has been disturbed by the fall of bombs and the crash of timber. copy has been destroyed by the effects of fire and explosion, proofs have been delayed by the emergencies of casualty surgery. Once again I must apologise the book has been out of print for many months. But although war creates difficulties it stimulates progress, and particularly the progress of surgery. This has been true throughout the ages. More suffering has been saved than was inflicted by all the wars of history and the conflict upon which we are now engaged has proved no exception. In former editions I was proud to claim the experience of nearly 50,000 civilian casualties but it would take more than a lifetime of civilian practice to equal the experience of the last few years. For example, whereas nearly all previous studies of fracture-dislocation of the talus have been based on isolated cases, it has been possible in the first two years of war, in the Royal Air Force Medical Service, to review no less than seventy five examples of this rare injury. Much has been learned and scarcely one page of the book remains untouched. I am grateful for these opportunities, and still more for the privilege of treating officers and airmen whose courage, endurance and spirit I have learned to respect. I would record my indebtedness to the Secretary of State for Air, Sir Archibald Sinclair, to the Air Council, and the Director General of Medical Services Air Marshal Sir Harold Whittingham. I am grateful to the team of surgeons who are working with me, many of whose contributions are acknowledged in the text, and particularly to Wing Commander Ian Dick and his wife who laboured so conscientiously in reading proofs and preparing the index. I owe a special debt to my faithful colleague and close friend Osmond Clarke, who was referred to by a grateful Czech pilot, with affection and only slight confusion, as Air Commodore Sir Nobby Clarke. His industry, enthusiasm and cheerfulness have been infectious; every orthopaedic specialist of the Service has gained by his inspiration, we have worked side by side, without his aid I could not have succeeded.

Every chapter has been redrafted. New sections have been added on open and infected fractures, war wounds, sequestrectomy, vascular injuries, immersion foot and shelter foot, traumatic oedema and the crush syndrome, gangrene due to tourniquets, Volkmann's ischaemic contracture, traumatic asphyxia and chest injuries, avascular necrosis of the hip joint, distraction of fractures, radiographic diagnosis of union, internal fixation of fractures, "no touch" technique, treatment by onlay grafting, burns and contractures of the hand. New recommendations are made in the treatment of supraspinatus tendon ruptures, Monteggia fracture dislocations of the forearm, recurrent dislocations of the shoulder, fractures of the navicular and fractures

of the spine Spinal injuries in air crews, naval casualties and air borne troops are reviewed New methods of treatment are described for congenital pseudarthrosis of the tibia, acromio clavicular dislocation, fracture dislocation of the talus and supracondylar fracture of the femur The technique of bone grafting the scaphoid is illustrated I am indebted to Mr Boyes for his aid in rewriting facio maxillary injuries to Mr Sol Cohen for his stimulus and interest in vascular surgery, and to Professor Seddon and his team at Oxford for material on ischaemic contracture I would pay special tribute to Mr Bremner Hight, a brilliant young surgeon of engaging personality whose clinical ability, operative skill and scientific achievement have been paid as one of the tragic costs of war His full contribution had not been made British surgery has suffered a grave loss



In 1943 it is possible to break your neck and enjoy it

In previous editions the final sentence read "The solution lies in the establishment of rehabilitation centres More can be done by a few weeks of rehabilitation than by months of light work or years of massage" The note was one of optimistic hope that an important development would be achieved But at that time it had not been accepted, I was still urging its merit I had urged it to a conference of surgeons in Liverpool in 1932, to the British Medical Association Committee on Fractures in 1934, to a meeting of important industrialists in London in 1935 to a joint committee of the B M A and T U C in 1936 and to the Delevigne Interdepartmental Committee in 1937 In company with Sir Walter Citrine and Dr Hill I outlined the details of a model rehabilitation centre, and after we had given evidence and been thanked as idealists whose heads were in the clouds the Committee turned to practical matters! But within a few months of the outbreak of war idealism became fact When an orthopaedic service was developed in R A F general hospitals throughout the country, rehabilitation

was introduced in every fracture ward and in four special rehabilitation centres. The Army followed suit with "hardening centres", centres are now being organised for the Miners' Welfare Commission, retraining centres are being established by the Ministry of Labour—rehabilitation is on every lip. It is no longer necessary to plead—rehabilitation has been accepted. Patients forget their broken backs, legs and arms. It is now possible to break your neck and enjoy it. Treatment is concentrated not only on the union of fractures but on the function of limbs, not only on surgery and manipulation but on gymnastics and recreation, not only on the relief of physical disability but on the cure of psychological disorder. This is the most striking development of fracture treatment in recent years, and a special chapter is devoted to its consideration.



These men have forgotten their broken backs, legs and arms

Shortly after the outbreak of war it became obvious that many surgeons, whose experience lay in other branches of surgery, would be engaged in the emergency treatment of bone and joint injuries. I held a series of courses at the British Postgraduate Medical School. In three years over 500 British, Dominion and American surgeons joined with me in discussions, lectures and practical classes, and in conclusion we indulged in a mental exercise—a ten minute review of the highlights of fracture treatment. At the request of friends I have made at these courses the review is included in this edition. It is intended to be no more than a cocktail and an appetiser.

"My story is not long but it took me a long time to make it short"—THOREAU. The new edition numbers over 200 pages more than earlier editions and for this reason it is published in two volumes, but it is no more difficult to read because the expansion is due largely to added radiographs, diagrams and photographs, time has again been devoted to brevity of text, careful choice of illustration and the difficult task of securing proximity between

relevant text and illustration. Indeed it has taken three times as long to illustrate the book as to write it, but I am convinced that this is the essence of good teaching. Through the Medical Research Council I have had invaluable aid from the Metal Box Company, who as a war contribution made available the services of Mr Hennell. His colour photography surpasses anything I have known. He has gained a wide knowledge of blood and bone, he has shared with me the vicissitudes of war, his camera and my scalpel have worked together.

To all who have helped I am grateful. To friends, colleagues and surgeons, to administrators, commanding officers and medical officers, to nursing sisters, nursing orderlies and technicians, to V A D clinical secretaries, medical gymnasts and masseuses, to Miss Hulme for long and loyal service, to my secretaries and radiographers for cheerful work in difficult circumstances, to Livingstones and Mr Charles Macmillan for skill in surmounting obstacles, to the engravers, printers and binders for care and attention to detail, to my wife Monica and Barrie for forbearance when "Daddy must work," to all who have co-operated I am most grateful.

Richard Owen Jones

LONDON

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PART I
THE PRINCIPLES OF
FRACTURE TREATMENT

CHAPTER I

THE REPAIR OF FRACTURES

A fracture of bone is the rupture of a living connective tissue, and its repair is achieved by the cellular growth which characterises repair in all living tissues. The training of a surgeon in the treatment of fractures must therefore begin with the physiological and pathological reactions of living tissues. Only after that does the art of surgery develop with the aid of mechanical apparatus, splints and plaster, and with the technique of manipulative and operative procedures.

HISTOLOGICAL FEATURES

Repair by granulation tissue—In its early stage, the histological picture of a healing fracture resembles that of any traumatic or inflammatory exudate undergoing organisation. The exudate is the partly fluid and partly clotted hæmatoma between the bone ends, beneath the raised periosteum, and in adjacent tissue spaces. Surrounding and invading the hæmatoma is a rapidly growing loose fibrous tissue of the cellular granulation type, showing evidence of hyperæmia in the dilated and engorged capillaries. Layers of granulation tissue formed by each fragment complete the resorption and organisation of the hæmatoma and if the fragments are immobilised in apposition both layers meet and unite.

Union by primary callus—Meanwhile trabeculae of hypertrophic cartilage cells developing in the marrow space and sometimes almost occluding the medullary cavity mark the first stage of callus formation by the endosteum (Fig. 1). Similar callus formation takes place beneath the periosteum, especially in younger patients where the membrane is easily stripped. The fibroblastic granulation tissue between the fragments, by which continuity has already been established, is then invaded and replaced by the irregular trabeculae of cartilage in which a proportion of bone cells and bone matrix gradually appears. Hypertrophic cartilage cells form the prominent feature of the histological picture until about the sixth week, but osteoblastic activity and matrix formation continue for a further three or four weeks. The earlier hyperæmia and vascular engorgement disappear, there is increasing calcification, and the exuberant growth of a formless, almost tumour like mass of irregular bone and calcified cartilage completes the first stage of bone union.

Consolidation by mature bone—Primary callus gradually matures to fully formed bone by a process of structural transformation. The irregular



FIG 1

Low power section of a repairing fracture. Vascular granulation tissue between the fragments is being invaded by irregular trabeculae of cartilage cells and new bone, arising mainly from the marrow and the endosteal and subperiosteal layers. Dead bone on the surface of the fragments, shown by empty lacunae, is undergoing absorption.

bone is replaced by lamellar trabeculae which are laid down in the usual lines of stress and strain. Periosteal new bone is absorbed and the original contour is restored. Tubulation is completed by reappearance of fat and marrow cells in the temporarily occluded marrow space. In a variable time, but on the average in about one year, the fracture may be so perfectly healed that its site cannot be determined.

CLINICAL FEATURES

The healing of a fracture is of course one continuous process. Three histological stages have been defined—repair by granulation tissue, union by primary callus and consolidation by mature bone—because they correspond to three stages in the clinical history of a fracture. Throughout the first stage the fragments are freely mobile and the delicate granulation tissue must be protected from the injury of movement and the injury of excessive traction. During the second stage the growth of cartilage and bone cells leads to increasing stability and the fragments become 'sticky'. Protection of the growing cells from the injury of movement by complete immobilisation of the fracture is still essential. The end of this stage may be judged by clinical tests: there is no longer elasticity or springing and the fracture is painless when strain is applied. This is the stage of *clinical union*. Splints and plaster may often be discarded. Nevertheless recalcification is far from complete and there may be no certain radiographic evidence of the union which has taken place. In the third stage of *consolidation of union* the bone is mature and fully recalcified and there is then radiographic as well as clinical evidence that the fracture is soundly united.

BIOCHEMICAL FEATURES

Deposition of calcium in the growing tissues constitutes one of the important distinctions between repair of fractures and repair of soft tissue injuries. During the first few weeks after a fracture there is a greatly increased concentration of calcium and phosphorus in the fracture hæmatoma. The general blood calcium level remains unchanged, and the local excess is derived from the bone ends. Three factors are concerned in this process of decalcification of the bone ends and hypercalcification of the hæmatoma.

1 **Histamine and acetylcholine**—In all injured tissues, the liberation of histamine and acetylcholine causes vaso dilatation and hyperæmia. Bone reacts to hyperæmia by decalcification¹ and transference of calcium from the fractured ends of the bone to the surrounding fluids continues until the hyperæmia subsides. Only then does recalcification begin.

2 **Phosphatase content of the fracture hæmatoma**—Within a few days the phosphatase content of the fracture hæmatoma, and of bone in the region of the fracture, increases to six or eight times the normal level.² Phosphatase is a bone enzyme, secreted by proliferating cartilage cells and osteoblasts, and always found where bone formation is most active.³ It liberates free phosphates by hydrolysis of the organically bound phosphoric acid of the

¹ Watson Jones and Roberts. Calcification, Decalcification and Ossification. *Brit Jour Surg* 1934 xxi 461.

² Botterell and King. Phosphatase in Fractures. *Lancet* June 1 1935 176.

³ Robinson. Significance of Phosphoric Esters in Metabolism. New York 1937.

plasma, thus causing supersaturation with calcium phosphate of the fluids bathing the bone¹. The phosphatase excess persists for about ten weeks, throughout the stage of active growth of cartilage cells and osteoblasts.

3 Acid tide of the fracture hæmatoma—During the first two weeks after fracture, the hæmatoma shows a marked acid tide, the pH then swinging back to the alkaline side of normal². The significance of this tide is not fully recognised, but it is clearly associated with local biochemical activity. The fracture hæmatoma may be regarded as a closed chemical factory, utilising its own store of calcium, mobilising, concentrating, and redepositing the salts as required. The process cannot be accelerated by attempting to raise the blood calcium level, by increasing the calcium intake, by alum treatment which controls the phosphorus intake,³ by vitamin therapy or endocrine therapy or even by the local implantation of calcium salts or phosphatase⁴. It is accelerated only by avoiding factors which prolong the period of decalcification. Mobilisation of calcium is an essential early phase of repair but it is a source of delay when unduly prolonged by the decalcification of disuse or the decalcification of traumatic and infective hyperæmia.

PATHOLOGICAL FEATURES

Hyperæmic decalcification of bone—Hyperæmia of bone is always associated with decalcification and ischæmia with sclerosis. In osteomyelitis, decalcification of bone continues so long as there is active infection and hyperæmia. When the hyperæmia subsides recalcification begins, and with the final reparative stage of fibrosis and ischæmia the bone undergoes dense sclerosis. Even if the bone itself is not infected, if it is adjacent to a focus of infection and comes within the field of hyperæmia it undergoes decalcification. This is seen typically in the septic finger where there is decalcification of the phalanges even if there is no bone infection. It is seen in certain tonsillar and pharyngeal infections where there is such a degree of hyperæmic decalcification of the atlas that the transverse ligament is loosened and the bone dislocates (p. 261). Rapidly growing vascular sarcomata cause bone decalcification even beyond the limits of the tumour which disappears when the blood supply of the tumour is reduced by intensive X ray therapy.

Traumatic hyperæmic decalcification—Similarly after injuries of bone, even after simple contusions traumatic hyperæmia causes local decalcification^{5,6}. If a joint is sprained the bone in the region of the damaged ligament is temporarily decalcified: the carpal bones of workmen who use pneumatic drills show many such foci. When there is an actual fracture, the reactionary hyperæmia causes decalcification of the bone ends which continues as long as the hyperæmia persists.

Traumatic hyperæmia of imperfect immobilisation—If the fragments are imperfectly immobilised shearing and twisting strains tear the young granulation tissue. Repeated traumatisations accounts for a recurring hyperæmia, and more and more of the bone ends undergoes decalcification. A crack becomes a cavity, a linear fracture becomes a gap fracture. This change is

¹ T. H. Man, Drummond McIntyre B. Gani, Phosphatase Activity in Early Callus. *Arch. Surg.* 1940 xl 43.

² Stirling. *Edin. Med. Jour.* 1932 xxxix 203.

³ Hellet. Parathyroid Function. Treatment by Aluminium Acetate. *Brit. Jour. Surg.* 1940 xxvii 631.

⁴ Bottorrell and King. Phosphatase in Fractures. *Lancet* June 1 1933 126.

⁵ Leriche and Fohrer. Physiology of Bone. London 1918.

⁶ Craig. Surgical Pathology of Bone. Edinburgh 1931. Hyperæmic Decalcification 227.

seen most clearly in fractures of the carpal scaphoid bone (Figs 2-4). Within a few days of injury the fracture may be so fine a crack as to be overlooked in radiographs. It may appear so trivial that there is a temptation to ignore it. If it is not immobilised perfectly the fine crack becomes an obvious fracture within two or three weeks and a cyst-like cavity within two or



FIG 2



FIG 3



FIG 4

A recent fracture of the scaphoid may be so fine a crack as almost to be overlooked (Fig 2). If it is not immobilised repeated traumata cause hyperæmic decalcification and the crack widens to a gap (Fig 3). When it is immobilised recalcification takes place and the fracture unites (Fig 4).

three months the bone ends showing concave surfaces. If at any stage the fragments are completely immobilised decalcification at once ceases, recalcification begins, the cavity slowly fills and the fracture unites.

If shearing movement continues indefinitely fibrous tissue is laid down parallel with the fractured surfaces and there is no continuity between the fragments. With the final stage of ischæmic fibrosis recalcification occurs not in a continuous mass of callus but in the plaque of bone across the concave bone ends. When the fractured surfaces become sclerosed non-union is established.

Infective hyperæmia and the repair of fractures—Whereas in a simple fracture which is protected from further injury traumatic hyperæmia subsides within about ten days in a compound infected fracture the initial traumatic hyperæmia is followed by infective hyperæmia which is even more



FIG 5



FIG 6



FIG 7

Infected compound fracture of the tibia two months after injury and two months after surgery. Provided that complete immobility is maintained the granulation tissue gradually recalcifies and the fracture unites.

intense and may persist for several months. Throughout this time decalcification of bone continues. Only when infection is controlled and hyperæmia subsides can recalcification begin. Treatment of the infection is obviously important. Sequestered fragments of dead bone must be removed as soon as they have separated otherwise persistent low grade infection delays recalcification and union of the fracture for months or even years. But important as early control of infection may be continued immobility of the fragments is no less important because decalcification due to the hyperæmia of repeated movement and strain is no less harmful in infected fractures than in simple fractures. Indeed the process of repair is the same and the need for complete immobility of the fragments is the same. The only difference between a simple fracture and an infected fracture is that in the presence of infection protection must continue for many months longer. If this is recognised non union of infected fractures is no longer a serious problem. Every infected fracture unites firmly by bone (Figs 5-7).

RATE OF REPAIR OF FRACTURES

Influence of age—In the average uncomplicated fracture there is continuity of granulation tissue in a few weeks, union by primary callus in two or three months, and consolidation of bone in four or five months. But many factors modify the rate of repair. Capacity for the growth of new tissue is greater in the infant than in the adolescent, and still greater than in the adult. A fracture of the shaft of the femur in a day old infant may be firmly united in one month, in a fifteen year old youth in two months, and in a fifty year old man only in three or four months. Malnutrition, cachexia, senile osteoporosis, and deficiency diseases may also exert a retarding influence.

Influence of type of fracture—In long oblique and spiral fractures where the marrow cavity is widely opened, there is a large vascular area to promote tissue growth, and union is usually more rapid than in horizontal fractures where medullary callus formation is more limited.

Delayed repair of gap fractures—Union is more rapid if the fragments are impacted into each other than if there is a gap between them. Given time, even considerable gaps may be bridged, but repair is more difficult. There is no natural fixation of the fragments, and absolute immobility is more difficult to achieve. Moreover if the gap is the result of continued traction, union is still more difficult. Any scar tissue subjected to traction becomes weak and attenuated, and in the case of a fracture, bone formation is discouraged. For this reason, the repair of fractures of long bones treated by excessive continuous traction is very slow^{1,2}. These factors, however, are responsible only for delay in repair. The fracture still unites even despite a gap between the fragments and despite continued traction, if complete immobility is maintained long enough.

Individual variations—Even in fractures of the same type, sustained by patients of similar physique and age, there may be wide variation in the rate of repair. To believe that repair is necessarily abnormal because it is slower than the average is a fundamental mistake. A fracture is not to be labelled "un-united" simply because union is incomplete within a certain number of weeks or months. It may be following a perfectly normal though slow progress.

VASCULARITY AND THE REPAIR OF FRACTURES

One of the most important factors in determining the rate of union is the vitality and vascularity of the fragments. If both fragments have a free blood supply, union is rapid. If the blood supply of one fragment is impaired, union is slow, if the blood supply of both fragments is impaired, union is very slow, and if one fragment is completely cut off from the circulation, union is very slow indeed.

Necessity for free blood supply—Rapid growth of the granulation tissue which initiates healing of a fracture demands a free blood supply, and even for the first few days an actual hyperæmia. The blood supply of normal bones is derived not only from the main nutrient vessels but from many other vessels entering the cortex through capsular, ligamentous and tendinous

¹ Fairbank. "Advances in the Treatment of Fractures."

² Watson Jones and Collart. "Slow Union of Fractures."

Post Grad Med Jour, 1937, xiii, 341.

Brit Jour Surg, 1943, xxx, 269.

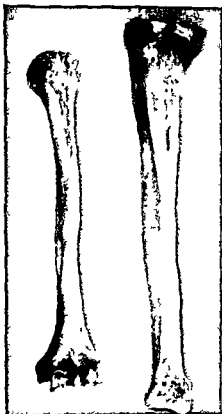


FIG 8

The lower shafts of the humerus and tibia are devoid of vascular foramina. The blood supply is entirely from the nutrient artery

of vascular foramina. The bone in these regions depends mainly for its blood on the nutrient artery (Fig 8). A fracture of the shaft may cut off this source of supply from the lower fragment, so that the vitality of this fragment is impaired, its contribution to the growth of granulation tissue and callus is feeble and union is slow (Figs 9 10). This difference in the vascularity of the two fragments may be a striking feature of operations performed for non union of fractures of the lower shafts of the humerus, ulna and tibia. Whereas the bone of the proximal fragment bleeds freely, the distal fragment may appear almost exsanguinated. In these cases the poor blood supply has been responsible for slow union and failure to prolong the period of immobilisation accordingly has been responsible for non union.

attachments. At the ends of long bones in the region of synovial reflections and joint capsules there are scores of vascular foramina. Through these foramina there pass not only veins from the bone but also arteries to the bone.¹ Indeed in growing children this metaphyseal area is more richly supplied with blood than any other region. Even in the adult the ends of long bones have a very free blood supply and fractures unite rapidly. Fractures of the neck of the humerus, supracondylar fractures of the humerus, fractures of the neck of the radius, Colles' fracture of the radius, supracondylar fractures of the femur, fractures of the tibial tuberosities and fractures just above the ankle joint are all characterised by rapid union. Similarly, the innominate bone which has many ligamentous attachments and which shows vascular foramina on all its surfaces, is freely supplied with blood and fracture repair is again rapid.

Impaired blood supply of one fragment

—On the other hand the lower third of the shafts of the tibia, the humerus and the ulna may be entirely devoid



FIG 9

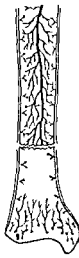


FIG 10

A fracture of the lower shaft of the tibia cuts off the blood supply of the nutrient artery from the lower fragment. Union is therefore slow.

¹ Johnson. Physiological Study of the Blood Supply of the Diaphysis. *Jour. Bone and Joint Surg.*, 1927, ix, 153.

Impaired blood supply of both fragments—Fig 11 is the radiograph of a double fracture of the shaft of the tibia. Both fractures were sustained simultaneously. The treatment of the two fractures by manipulative reduction and immobilisation in an unpadded plaster cast was identical. Nevertheless the rate of repair is very different. The upper fracture was firmly consolidated in five months. Fig 12 shows the radiograph after eleven months of continuous immobilisation. Repair of the upper fracture is so complete that practically all trace of the injury has disappeared, and yet the lower fracture is still far from firmly united. What is the difference in the blood supply of the two fractures? The upper fracture has a normal blood supply on the proximal side and an impaired supply on



FIG 11

Double fracture of the lower shaft of the tibia. At the upper fracture the blood supply of one fragment is impaired. At the lower fracture the blood supply of both fragments is impaired.



FIG 12

Union at the upper fracture was somewhat slow and was complete only after five months. Union of the lower fracture is very slow. It is still incomplete after eleven months.

the distal side which has been cut off from the nutrient artery. Union therefore was rather slower than normal. The lower fracture has a poor blood supply on both sides, even the proximal fragment has in this case been deprived of its blood by a second fracture higher in the shaft. Since neither fragment has an adequate blood supply, neither is capable of promoting vigorous granulation tissue growth and a bone grafting operation was necessary before firm union was secured.

Complete loss of blood supply to one fragment—If the blood supply is entirely cut off from one fragment so that it is completely avascular, the fragment remains inert and can take no part in the process of repair. This occurs in certain fractures of the neck of the femur, the carpal scaphoid and other bones (see Chapter V, Avascular Bone Necrosis). There is radiographic evidence of this avascularity: for the dead bone cannot decalcify and it appears more dense than the adjacent living bones which undergo

disuse and hyperemic decalcification. If one fragment takes no part at all it is obvious that repair must be extremely slow. The relative indolence



FIG 13



FIG 14



FIG 15



FIG 16



FIG 17



FIG 18

Dislocation of lunate and half scaphoid with complete loss of blood supply to the proximal scaphoid fragment (proved by its apparent density—see p 75). Despite this radiographs two months six months ten months fourteen months and eighteen months after injury show steady invasion of the avascular fragment and ultimately union of the fracture.

of cartilage and bone cell proliferation is shown by absence of the local phosphatase excess¹ which is characteristic of all other types of fracture (p 5). But although repair is slow it can still be accomplished if immobilisation is suitably prolonged. The living fragment promotes tissue growth which slowly invades and ultimately replaces the dead bone. The process may occupy eighteen months or even two years and immobility of the fragments must be maintained throughout this time (Figs 13-18).

CHAPTER II

DELAYED UNION AND NON-UNION

Non union of fractures is due to the failure of surgeons much more than to the failure of osteoblasts. With few exceptions it is an avoidable complication. It has been customary to enumerate many etiological factors such as imperfect apposition of fragments, interposition of soft tissues, distraction of the fragments, impairment of blood supply, functional disuse, infection of bone, osteoporosis, senile change, operative interference, stripping the periosteum, plugging the medulla, reaction to plates and screws, the inhibitory effect of synovial fluid and lack of blood clot between the fragments. The latest addition to the list is compression of the fracture hæmatoma by an unpadded plaster cast. But these conditions although they may influence the rate of union are not the cause of non union. There is only one cause of non union of fractures with a continuous hæmatoma between the fragments—the cause of non-union is inadequate immobilisation.

The rate of repair has already been discussed. Age makes the difference between union of a fractured femur in three or four weeks in the infant, and three or four months in the adult, but at all ages, even over the age of ninety, fractures of the femur unite. Impairment of blood supply may delay the process of repair, but if fractures showing this delay are protected long enough they still unite. Operative exposure of a fracture may reduce the blood supply by stripping soft tissue attachments from the bone, but if post operative immobilisation is adequate, plating and other operations do not cause non union. Excessive traction causes striking delay in the healing of fractures, but union still takes place if immobilisation is prolonged. Even severe infection of a fracture is not a cause of non union if immobilisation is maintained for twelve months or even longer.

Age, constitution, blood supply, infection, type of fracture and method of treatment make union difficult or easy, slow or rapid. If these conditions are favourable, as for example in fractures near joints where bone is vascular and cell growth vigorous, a fracture may unite despite imperfect mobility. But if the conditions are unfavourable and cell growth is slow and difficult, the fracture unites only when immobilisation is adequate and prolonged. The cause of non union in gap fractures, infected fractures, plated fractures and fractures with poor blood supply is not the gap, the infection, the plating or the blood supply, but the failure to recognise that these factors cause slow union, and the failure to prolong immobilisation accordingly.

In a series of 800 consecutive fractures of the shafts of the femur and tibia recently investigated,¹ there was a high proportion of comminuted, contaminated and severely infected fractures, many different methods of treatment had been used including manipulation and plaster, skeletal traction, open reduction, internal fixation, etc. Whatever technique had been employed, there was always insistence on continued and uninterrupted

¹ These cases are analysed in detail in Chapter XXXI.

immobilisation until repair was complete. In the whole series *there was not one case of non union*. Many fractures were slow in uniting but not a single fracture failed to unite.

DIFFERENTIATION OF SLOW UNION, DELAYED UNION AND NON-UNION

Formerly, a certain number of weeks was fixed as the period required for the union of a fracture in each region—clavicle three weeks, scaphoid four weeks, tibia eight weeks, femur ten weeks, and so on. More recently the periods have been extended. Three months is now granted, or even four months,¹ but the fundamental error is still made of fixing a period at all. The dictates of a calendar are accepted, and every fracture not united within the specified time is labelled "un united." A new regime of treatment is then instituted: the plaster is removed, heat and massage are employed,



FIG 19



FIG 20



FIG 21

Slow union

Fracture of the humerus three, six and twelve months after injury. There is indolence and slow union, but no cavitation or non union.

hammering and darning² are prescribed. A walking calliper splint is fitted. Immobilisation is necessarily suspended. It would be as reasonable for a gardener to uproot every plant not in flower by a specified date in order to stimulate its growth by heat and chemical. Growth may have been naturally slow, or it may have been delayed by injudicious treatment. A delay of weeks or even of months does not mean that union must necessarily fail. Whatever the passage of time, if a fracture is still in the stage of slow union or delayed union, the essential treatment is to leave it undisturbed and protect it by continued immobility.

Slow union—The fracture line is clearly visible, but there is no undue separation of the fragments, no cavitation of the surfaces, no decalcification and no sclerosis. These are the appearances of any fracture during the first few weeks, if the appearances are maintained for more than a few weeks, union is slow (Figs 19-21). Indolence of the fracture is due to

¹ T. P. McMurry, Delay in the Union of Fractures. *Brit. Med. Jour.* 1912, 1, 2.

² Hugh Owen Thomas's "Lining and darning" consisted of percussion with a rubber hammer and passive venous congestion by means of a tourniquet repeated two or three times a day.

its type, its blood supply, or the age and constitution of the patient Union has not been unnecessarily delayed, and it is not an un united fracture It will still unite if it is immobilised long enough

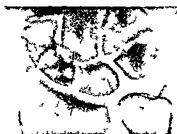


Fig 22



Fig 23

Delayed union

Six months old fracture of the scaphoid showing decalcification and cavitation (Fig 22) After adequate immobilisation the decalcification ceases and the fracture unites (Fig 23)

Delayed union—Repeated movement of the fragments with reactionary hyperæmia has decalcified the bone ends, and the fracture line has widened to a "cavity" with an outline woolly and ill defined As yet there is no recalcification or sclerosis (Figs 22 23) It is a case of delayed union, not of non union Repair is not yet complete, the gap is filled with granulation tissue not with scar tissue The essential treatment is to avoid the movement and strain which has been responsible for decalcification The fracture must be more completely immobilised and the immobility must be continued for many weeks or months until losses are made good until the gap is recalcified and union consolidated

Established non-union—If movement of the fragments still continues the third stage develops after many months The gap between the fragments may have partly filled but the margin is now well defined The fractured surfaces are relatively smooth, and when recalcification causes actual sclerosis the battle is over cellular activity ceases and non union is established (Fig 24) The fragments are joined by dense scar tissue, and a false joint may develop with serum loculated in the cicatricial mass No matter how long immobilisation of such a fracture is continued, bony union will never occur It is essential first to break down the sclerosed tissue, and to initiate a new traumatic hyperæmic decalcification by cutting through vascular scar and dense bone, thus exposing vascular tissues capable of promoting



Fig 24

Established non-union

Old fracture of the tibia showing dense sclerosis at the fractured surfaces Non union is now established

granulation tissue growth. In fact it is essential to reconstruct the conditions of a recent fracture and then to immobilise adequately and continuously as should have been done in the first place. These objects may be achieved by freshening the fractured surfaces or by drilling the fragments in many directions through a short incision.¹ They may be achieved still more certainly by a bone grafting operation because cutting a bed removes sclerosed bone effectively and the fitting of a graft augments immobilisation.

SLOW UNION DUE TO POOR BLOOD SUPPLY

The slow union which sometimes occurs in otherwise uncomplicated fractures is illustrated in Figs 28-32. A healthy middle aged man sustained a fracture of the shafts of both leg bones. There was a punctured wound which healed without infection. No open operation was needed. No continuous traction was employed. After ten days in a padded plaster cast,

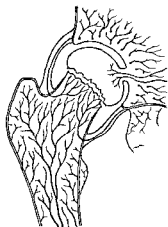


FIG 28

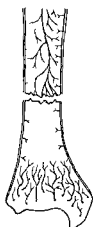


FIG 29

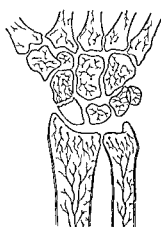


FIG 30

Impairment of blood supply of one fragment may cause slow union in fractures of the neck of the femur waist of the scapula neck of the astragalus and certain fractures of the shafts of long bones.

complete immobility was maintained by an unpadded plaster from the toes to the groin. Radiographs taken after three months show indolence of the fracture and the patient was warned that progress would be slow. A bone grafting operation was considered with the object of accelerating union but the patient preferred conservative treatment with its equally certain result. Functional treatment was maintained by active exercises and weight bearing in plaster. At the sixth month there was still no evidence of calcified callus and clinical tests showed unsound union. Nevertheless confidence remained unshaken and immobilisation was continued. At the ninth month the fragments began to fuse and after twelve months' continuous immobilisation union was sound. If immobilisation had ceased at an earlier date the fracture would have failed to unite. Slow union would have become non union. The non union would not have been inevitable as so often is assumed. It would have been avoidable.

Slow union of this degree is seldom seen in fractures of the shafts of long bones not complicated by infection, excessive traction or open operation.

This is described as Beck's bone drilling. In Beck's article in 1909 was preceded fifty-five years by Wolff's 'United Fractures Treated by Long Axial Drilling of the Bone Ends' *Brit Jour Surg* 1914 II 43.



FIG 28
Three months



FIG 29
Six months



FIG 30
Eight months



FIG 31
Nine months

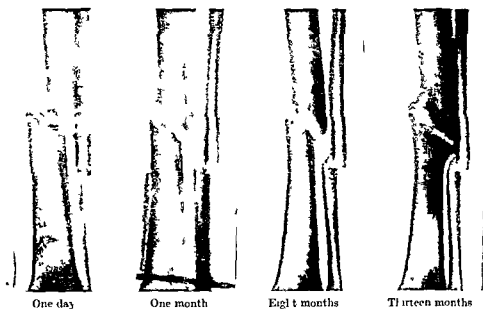


FIG 32
Twelve months

Fracture shaft of tibia with slow union due to poor blood supply of distal fragment showing the degree of union on three six eight nine and twelve months after fracture. Prolonged and complete immobilisation is essential to avoid non union.



FIGS 33-37—Distraction due to excessive pull was corrected by the twentieth day, but the harm had been done. There is typical indolence and slow union.



FIGS 38-41—Distraction continuing for many weeks is still more serious. In this case there has also been inadequate immobilisation as shown by heaped up bone round the fracture.

but it is significant that the normal process of repair can sometimes last as long as twelve months. In fractures of the carpal scaphoid bone and fractures of the neck of the femur impairment of blood supply occurs more often (Figs 25 27). An accurate forecast of the necessary period of immobilisation cannot be made. The fracture of the scaphoid with complete interruption of blood supply illustrated in Figs 13 18 united only after eighteen months. Similarly in fractures of the neck of the femur experience recently gained by the nailing operation shows that the process of repair may occupy from twelve months to two or three years.

SLOW UNION DUE TO EXCESSIVE TRACTION

Slow union of fractures has occurred with increasing frequency in recent years. There can be little doubt that this is associated with the higher standard of accurate reduction and end to end apposition now demanded and particularly with the methods of skeletal traction which are employed in achieving this reduction. The danger does not lie in the correction of overriding of bone fragments by neutralising the elastic recoil and spasm of muscles but in over correction in excessive pull which separates the fractured surfaces and distracts the fragments and especially in over correction which is maintained for several days or weeks by the suspension of heavy weights from a skeletal traction pin.

The danger of skeletal traction.—Powerful traction is available from a pin driven through the bone and attached to a weight suspended over a pulley at the foot of the bed. By increasing the weight to 20 or 30 lb it is easily possible to cause overlengthening or distraction. In compound fractures especially in war injuries where the muscles surrounding the bone are severely damaged muscle weakness may be so profound that distraction is produced by as little as 10 to 15 lb weight. Moreover there is often such loss of tone that when the fragments are once distracted they are not pulled together again even when the weight is reduced and the gap persists. The bridging of such a gap offers unnecessary difficulty in the repair of a fracture. But the delay is much greater than would be accounted for by the time taken in bridging a gap. Many months are added to the necessary period of immobilisation even by slight separation which is rapidly corrected (Figs 33 37). If the fragments are distracted at the most dangerous period not within a few



FIG 40
Ten weeks



FIG 43
Fifty weeks

Compare the rapid union of spiral and oblique fractures (similar to those in Figs 33 41) when no traction is employed and red displacement is prevented by a vertical transfixion screw (Operative Wound and Fracture).

days of injury but after several weeks when the fracture hematoma is organising and being replaced by cellular tissue a fracture which would otherwise unite in two or three months unites only after ten or twelve months. It behaves exactly as if the blood supply had been seriously reduced as in the case shown in Figs 28 3^o. It is probable that this in fact is what occurs and that slow union after excessive traction is due not only

to the obvious tearing apart of young cellular layers but also to the strangling by tension of capillaries in the organising hematoma so that indolence continues even after distraction is corrected (Figs 33 41). It is noteworthy that the effect is most pronounced in the middle third of the tibia where the blood supply is already poor and it is less marked in a vascular bone like the femur. The delaying influence is least evident in the shaft of the fibula.

The greater danger of traction without immobilisation—There is even greater danger when traction is used to control alignment as well as to prevent overriding the method adopted by Bohler in treating fractures of the shaft of the femur and by some surgeons in treating fractures of the tibia. No local splintage or plaster is used the limb rests on a simple cradle and the weight alone maintains length alignment and apposition. It is often found that weight sufficient to prevent angulation causes distraction of the fragments and if the weight is reduced to prevent distraction angulation recurs (Figs 45 49). The temptation to employ excessive traction is almost irresistible. Moreover to the delay of excessive traction is added the delay of inadequate immobilisation. This technique has been responsible for many cases of non union

particularly in fractures of the shaft of the femur. Skeletal traction must be used with caution. The practice of attempting gradual reduction of displacement over a period of many weeks by continued heavy traction on the limb must be abandoned. The object of treatment should be to correct displacement at once within a day or two of injury and then to immobilise in the corrected position using the least possible traction and certainly no more than is strictly necessary to prevent redisplacement. In the tibia it is better to prevent redisplacement of an unstable fracture by a vitalium transfixion screw (Figs 4^o 43) than by a traction pin and suspended weight.

The danger of skeletal transfixion—Another technique recently developed



FIG 44

An example of grossly excessive tibial traction. Not only as the limb overlengthened and non seriously delayed but the knee joint ligaments were severely stretched and the cruciate ligament was actually a ulcer with a fragment of bone



FIG 45
Six weeks



FIG 46
Twelve weeks

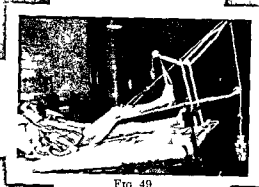


FIG 49



FIG 47—Fourteen weeks



FIG 48—Fourteen months

Excessive pull continuing for six weeks caused distraction of the fragments (Fig 45). Even when the weights were reduced this was not corrected (Fig 46). Still further reduction of the weight allowed angulation to recur (Fig 47). Finally a bone grafting operation was necessary (Fig 48). Excessive traction without immobilisation causes slow union and recurrent deformity if continued it causes non union (Bone graft by Wing Commander Armstrong)

in the United States, is to drive one or more pins through each fragment, reduce the displacement by mechanical means and incorporate the pins in a plaster cast, or in a special apparatus which locks them together and prevents approximation (Figs 288 290) If the fragments are fixed in distraction, with a slight gap between the fractured surfaces union is very slow The sequel is exactly similar to the slow union of excessive skeletal traction

DELAYED UNION DUE TO INADEQUATE IMMOBILISATION

Two purposes are served by the immobilisation of a fractured bone (i) control of position thus avoiding displacement of the fragments and mal union and (ii) protection of growing cells, thus avoiding delayed union and non union Splints or plaster which are adequate for the first purpose may yet be inadequate for the second. All the injuries illustrated in Figs 30 32 may appear to be adequately immobilised The fracture of both bones of the leg is protected from angulation and deformity by the plaster cast to the knee and still more certainly by the plaster to the mid thigh but neither of these plasters controls rotation movement of the upper fragment of the tibia Even the mid thigh cast allows rotation of the femur to be transmitted to the upper fragment of the tibia, because the knee joint is fully extended Unless this rotation movement is prevented delayed union or non union will result The short forearm plaster may prevent gross displacement of the fracture of both forearm bones (Fig 52) but the cast is not controlling supination and pronation movement, and this movement is accompanied by rotational strain on the fracture of the ulna which causes non union Neither the cock up splint nor the plaster cast used for the fracture of the scaphoid is fully controlling wrist movement (Fig 50), and although the fragments of the scaphoid will not displace, the fracture will certainly fail to unite At one time the degree of immobilisation illustrated in these figures was accepted as classical treatment At that time non union was so common that lists of causes were enumerated to explain it Now that the need for protection of growing cells by more complete immobilisation is recognised non union of these fractures does not occur

Protection of the growing cells demands (i) adequate immobilisation, (ii) uninterrupted immobilisation (iii) immobilisation for an adequate period (iv) complete immobilisation until union is sound

Adequate immobilisation—As a rule the joints above and below the fracture must be immobilised even although less complete fixation would suffice to prevent displacement The special precautions needed to protect fractures from rotational and shearing strains are discussed in later paragraphs

Uninterrupted immobilisation—The immobilisation must be continued without interruption If the only object was to prevent deformity, movement of the fragments at the time that a plaster is being changed would be unimportant so long as the position was finally adjusted Actually every single movement or strain sets back the process of repair If a new plaster is to be applied there must be no interval when unguarded movements are permitted The discarded plaster must be removed by the surgeon himself in the theatre not by a nurse in the ward or an assistant in the ante room The surgeon must support the limb protect the fragile tissues and never permit careless handling and strain which may even amount to refracture



FIG 50

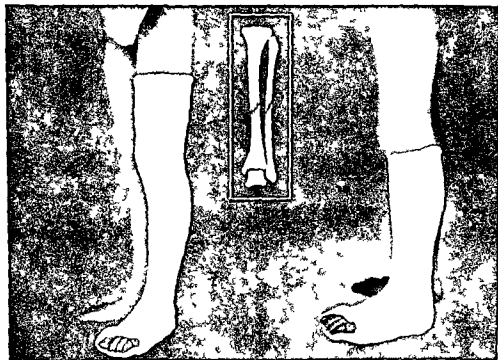


FIG 51

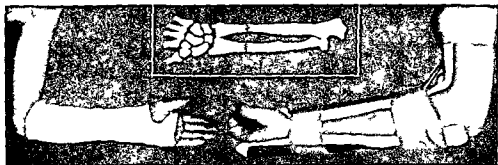


FIG 52

The cause of non union of fractures

FIG 50—Fracture of carpal scaphoid bone FIG 51—Fracture of shafts of tibia and fibula
FIG 52—Fractures of shafts of radius and ulna

In every case the immobilization is inadequate. Although the splints and plaster used may suffice to prevent deformity they do not completely protect the growing cells, and delayed union or even non union will result.

It is obvious that union of a fracture can never take place if refractures are sustained at regular monthly intervals every time the plaster is changed.

Immobilisation for an adequate period—The immobilisation must continue until the fracture is united however long that may be—eight to twelve weeks in the average case four months in some cases and six to twelve months or even longer in exceptional cases. There is no fixed number of weeks for the repair of any fracture. If every fractured tibia is taken out of plaster at the end of eight weeks all slowly repairing fractures fail to unite. If the arbitrary period is fixed at ten weeks or at twelve weeks the incidence

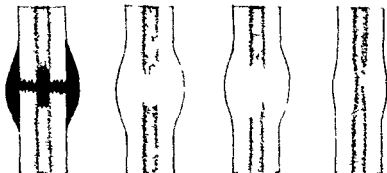


FIG 53

Healing of a perfectly immobilised fracture. The fragments are joined by an uninterrupted growth, in the early stages of granulation tissue and in the later stages of callus. Continuity is established.

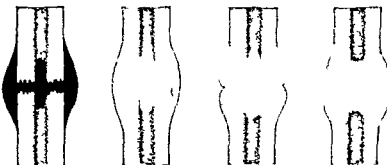


FIG 54

Healing of an imperfectly immobilised fracture. Shearing and rotation strains create a plane of cleavage. Continuity is interrupted and fibrous tissue is laid down parallel with the fractured surfaces.

of non union is lower but there will still be failures. Immobilisation must continue regardless of time until there is clinical evidence of sound union.

Complete immobilisation until union is sound—It is not enough to immobilise a fracture completely for eight or ten or twelve weeks and then protect it by some less adequate form of immobilisation—for example in a fracture of the tibia by changing from a full length plaster to a below knee cast. If union is already sound such protection is unnecessary. If union is not already sound the inadequate immobilisation induces traumatic hyperæmic decalcification once more and callus which has begun to calcify undergoes decalcification and absorption (see Figs 60-63).

The danger of rotation and shearing strain—The type of movement most

harmful to the repairing fracture is a rotational or shearing strain which creates a plane of cleavage between the fragments (Figs 53 54). Fibrous tissue is laid down in the plane of movement, parallel with the fractured surfaces. When recalcification finally takes place, there is no continuous mass of callus to be calcified, and the sclerosis merely seals off the bone ends. This type of rotational strain is clearly evident in all the regions where non union was so common in former days—the lower shaft of the ulna, the lower shaft of the humerus, the lower shaft of the tibia, the waist of the scaphoid and the neck of the femur.

Rotation strain in fractures of the shaft of the ulna—When fractures of the shafts of both forearm bones were treated by a pair of gutter splints from the level of the hand to just below the elbow, the fracture of the radius usually united without difficulty, but the fracture of the ulna remained



FIG 53

Un united fracture of the ulna

un united (Fig 55). The splints prevented angulatory movement of the fragments, but they did not prevent pronation and supination of the forearm. This movement is a bucket handle swing of the radius round the fixed axis of the ulna. The two fragments of the radius swing together and there is no torsional strain, the fragments of the ulna rotate on each other and the torsion prevents union (Figs 56 57). So common was this error of treatment that fractures at this level were regarded as peculiarly susceptible to non union. Indeed this was chosen as the site of election for arthroplasty of the forearm in cases of radio ulnar ankylosis. It was believed



FIG 56



FIG 57

A forearm plaster does not prevent supination pronation movement and the resulting torsion of the fragments of the ulna causes non union

that if the lower shaft of the ulna was divided by osteotomy, the formation of a false joint was almost inevitable. Actually if the forearm is properly immobilised by a plaster cast from the hand to the upper arm, so that radio ulnar movement is completely arrested, fractures of the lower shaft of the ulna always unite firmly.

Union is often slow. It may be necessary to continue immobilisation for four months or even longer, and the complete plaster to the upper arm must be retained throughout this time. The surgeon must not be tempted by the fear of elbow stiffness into replacing the complete plaster by a short forearm cast at the eighth or tenth week. A fracture of the ulna is subjected to greater rotational strain in a short forearm plaster than if it is in no plaster at all. Movement at the superior radio ulnar joint is not limited by such a plaster and pronation and supination are still possible, but close moulding of the plaster round the wrist obstructs movement at the inferior radio ulnar joint. Rotation occurs more easily at the unsoundly united fracture of the ulna than at the stiffened inferior radio-ulnar joint. The fracture of the radius may go on uniting because there is no torsional strain, but

recalcification at once ceases at the fracture of the ulna. Decalcification takes its place, callus already developed is absorbed and non union results.



FIG 58

Most fractures of the shaft of the humerus show vigorous callus formation and rapid union despite the obviously incomplete immobilisation of simple gutter splints

formation that union takes place within five or six weeks even despite incomplete immobility. Short gutter splints or a plaster slab are sufficient

and if the wrist is suspended from the neck by a collar and cuff sling, mild traction is maintained by the weight of the limb (Fig 58). Occasionally, however, fractures in the lower shaft are of exactly the opposite type. Clinical tests at the sixth week show that the fragments are still freely mobile and union has scarcely begun. These are examples of slow union due to injury of the nutrient artery and impairment of the blood supply. The same slow union may result from excessive traction. The surgeon must be on guard. The short splints and sling which are adequate for other fractures of the humerus are entirely inadequate for the indolent fracture. If they are relied upon the fracture will fail to unite (Fig 59). Every movement of the forearm and hand away from the chest wall rotates the lower fragment of the humerus and strains the cellular growth which is already so feeble. Firm union is secured only if the fracture is

Freedom from shearing and rotation strain in fractures of the ribs—The ease with which fractures of the ribs unite despite constant respiratory movement was claimed to support the old fallacious view that slight movement encouraged union. But respiratory movement of the ribs is exactly analogous to the bucket handle swinging of the radius. Both rib fragments move together, and the intercostal fibrous and muscular tissues provide a perfect natural protection from shearing and rotation strain.

Rotation strain in fracture of the shaft of the humerus—A fracture of the shaft of the humerus is completely immobilised only if both elbow and shoulder joints are fixed by means of a plaster spica (Chapter XXIII). Nevertheless most fractures of the humerus are characterised by such vigorous callus



FIG 59

Indolent fractures of the shaft of the humerus with slow union will not unite at all if immobilisation is incomplete. Both shoulder and elbow joints must be completely immobilised by means of a plaster spica.

strains the cellular growth which is already so feeble. Firm union is secured only if the fracture is

protected from all rotation strain by the complete immobilisation of a plaster spica

This complete immobility must sometimes be continued for many months. The harmful effect of even delayed rotatory movement is proved by the case shown in Figs 60-63. Union was characteristically slow. Despite complete immobility of the fragments consolidation was not complete by the fifth month. It was then believed that the fracture was firm enough to begin shoulder exercises and an unpadded plaster cast was applied from the wrist to just below the shoulder joint. Within two months the effect of the rotation strain is obvious. Union is breaking down, the callus has decalcified and absorbed and established non union is already threatening in the

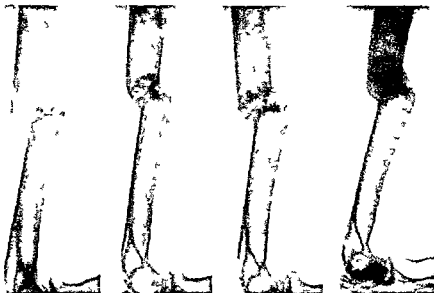


FIG 60

FIG 61

FIG 62

FIG 63

Fracture shaft of humerus three five seven and twelve months after injury. After five months immobilisation, union was almost complete (Fig 61). An unpadded plaster to the axillary level was applied to allow shoulder exercise. Rotation of the fragments within the plaster caused absorption of callus and two months later non union was threatening (Fig 62). It was avoided only by reconstituting complete immobility for four months longer.

tendency to sclerosis of the fractured surfaces. Shoulder movements were stopped, complete immobilisation was reconstituted by a plaster spica and after several months of further fixation consolidation was complete.

A plaster cast from wrist to axilla in the treatment of an indolent fracture of the humerus is comparable to a below-elbow plaster cast in the treatment of an indolent fracture of the ulna. In each case the half plaster is more dangerous than no plaster at all.¹

Rotation strains in fractures of the shaft of the tibia—Fractures of the shaft of the tibia show the same response to rotation strain. The fracture is not immobilised unless both knee and ankle joints are immobilised. A plaster to the knee may control general alignment, but it does not immobilise

¹ The hanging cast technique advocated by Caldwell (J. Amer. Surg. Ass. 1914) is based on the same principle as the simple gutter splints and collar-and-cuff splint shown in Fig. 58, but plaster is used from the wrist to the axilla as described in the case quoted above. While adequate for all fractures of the shaft of the humerus in which repair is vigorous and rapid, this technique is obviously dangerous for indolent fractures and will cause non-union. In such cases the elbow joint must be immobilised as well as the elbow.

the fracture because twisting of the knee can rotate the upper fragment of the tibia within the plaster. The plaster can be twisted round the leg. Close moulding round the foot and ankle gives a secure grip of the distal fragment of the tibia and every twisting movement of the plaster is transmitted directly to the site of fracture. The position is exactly similar to the use of a short forearm plaster for fractures of the ulna or a wrist to a wrist plaster for fractures of the humerus. *Half a plaster is more dangerous than no plaster.* The below knee cast has no place in the treatment of tibial fractures even in the later stages. It gives a false sense of security. Either the fracture is already united and the plaster is unnecessary or the fracture is not united and the plaster is harmful. The plaster must extend from the foot to just below the groin. Moreover the knee joint must be slightly flexed. Even a full length cast does not perfectly immobilise a fracture of the tibia if the knee joint is fully extended. The terminal degrees of extension are accompanied by a locking movement which binds the femur and tibia together and makes them rotate together. Rotation of the femur twists the upper fragment of the tibia and the torsional strain may prevent union. When the fracture is in the upper shaft of the tibia it may even be advisable to include the hip joint in a plaster spica so that there is no possibility of rotation of the femur.

Rotation and shearing strain in fractures of the scaphoid.—It is not many years since a world famous radiologist said that he had never seen a fracture of the scaphoid unite by bone. This was the day when the accepted treatment was the application of a cock up splint for a few weeks (Fig. 50).

Such a splint does not protect from shearing strain. The scaphoid lies half in the proximal row and half in the distal row of the carpus (Figs. 64-65). With lateral movements of the joint the distal fragment tends to move with the distal carpal bones and the proximal fragment with the proximal bones. This shearing strain must be controlled by a plaster cast extending round the sides of the wrist and hand thus preventing adduction and abduction movement.



FIG. 64



FIG. 65

A fracture of the waist of the scaphoid coincides with the line of the mid carpal joint and even slight wrist movement causes shearing of the fragments.

Is shearing the only strain responsible for the former frequency of non union

in fractures of the scaphoid? In the other sites we have discussed—the shaft of the ulna, shaft of the humerus and shaft of the tibia—it was rotational strain which was mainly responsible. Analysis of wrist movement shows that imperfect immobilisation allows similar rotational strain in fractures of the carpal scaphoid bone. Dorsiflexion and palmar flexion are not simple up and down movements of the hand. The shape of the articular surfaces shows that this movement consists of a screwing of the proximal row of the carpus into the radius and of the distal row of the carpus into the proximal row. Examine your own wrist first at rest and then gripped in dorsiflexion. Dorsiflexion is accompanied by definite supination of the carpus and hand on the forearm bones and this screwing movement locks the dorsiflexed wrist in the position of strength. When the scaphoid is fractured one thread of the screw is stripped. The slightest

dorsiflexion or palmar flexion movement screws the fragments on each other causes rotational strain tears the young granulation tissue produces traumatic hyperæmia and decalcification and causes delayed union. It is obvious that a plaster cast with padding is inadequate. Every degree of movement is harmful and closely fitting unpadded plaster casts are necessary. If this immobility is maintained uninterruptedly all recent fractures of the scaphoid unite by bone and since in most cases the blood supply has not been damaged union is secured in about eight weeks. If the plaster is allowed to become loose to crack or to become wet and sodden and it is not immediately replaced union is always delayed the fracture may then unite only after ten or twelve weeks. If the plaster is occasionally removed for clinical and radiographic examination and the wrist is left unprotected for several hours or days union is still more delayed and if these interruptions are repeated often enough delayed union passes finally into established non union.

Shearing and rotation strains in femoral neck fractures—Until recent years fractures of the neck of the femur were treated in plaster and non

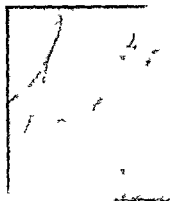


FIG 66

Fracture of the neck of the femur treated in plaster. Non union is due to twisting movements at the site of fracture.



FIG 67

Fracture of the neck of the femur treated by nailing. The fracture has united because of absolute immobility.

union occurred in about 50 per cent of cases. The modern treatment is to fix the fragments by a three flanged nail over 90 per cent of cases unite. What is the difference between the fixation of plaster and that of a three flanged nail? In a plaster splint slight twisting and turning of the pelvis and therefore of the loose femoral head is still possible within the plaster. There is continued shearing and rotation movement of the fragments. On the other hand a three flanged nail affords complete fixation. The length of the nail prevents shearing and the flanges prevent rotation of the fragments. If the nail is perfectly placed so that there is absolute immobility of the fragments union is possible in almost every case.

The influence of shearing strain is proved by comparing the two types of femoral neck fracture. The common type is an adduction injury the shaft and neck of the femur are adducted in relation to the head (Fig 68). The less common type is an abduction fracture the shaft and neck are abducted on

the head (Fig 69) In the adduction fracture weight bearing shears the fragments on each other even if there is no weight bearing and the patient lies in bed, contraction of the hip muscles causes similar shearing The line of movement of the fragments is across the fractured surfaces, the fragments rub on each other there is hyperemic decalcification and the bone is worn away The whole femoral neck may disappear These are the fractures which never unite spontaneously which seldom unite in a plaster spica and which require the greater fixation of a nail The less common abduction fractures are displaced in the opposite direction, and the effect of weight bearing and muscle retraction is to impact the fragments together The axis of



FIG 68

Adduction fracture neck of femur

The axis of movement of the fragments due to muscle retraction and weight bearing passes across the fracture There is shearing strain The fracture does not unite unless perfectly immobilised



FIG 69

Abduction fracture neck of femur

The axis of movement of the fragments due to muscle retraction and weight bearing passes through the fracture There is no shearing strain The fracture unites even without treatment

movement is not across the fracture causing shearing but through the fracture causing *impaction* These fractures always unite, even without treatment

The proof that shearing strains cause non union would be complete if we found that the tendency of the adduction fracture to non union became a tendency to union when the adduction displacement was changed to an abduction displacement We find that this is so If such a fracture is treated by a trochanteric osteotomy, and the shaft is abducted so that the line of weight bearing and of muscle traction now passes through the fracture instead of across it the fracture promptly begins to unite (Figs 70 71) This fact has an important bearing on the treatment of delayed union in old untreated fractures of the femoral neck (Chapter XXVIII)

Shearing and rotation strains after plating of fractures—Screws and wire and particularly bone plates are said to be responsible for non union It

has been suggested that a slight grip between the fragments is maintained by the rigidity of the plate but we know that much wider gaps than this can be bridged. Again it has been suggested that the chemical reactions of the metal interfere with the normal acidity of the fracture hæmatoma, yet we know that nails driven through the middle of the hæmatoma do not prevent union of fractures of the neck of the femur. Paradoxical as it may seem the non union so often associated with bone plating is again due to inadequate immobilisation. At the time of operation the fragments are perfectly immobilised by the metal plate and screws¹ so firmly indeed that the limb may be moved in every direction with apparent safety. The surgeon

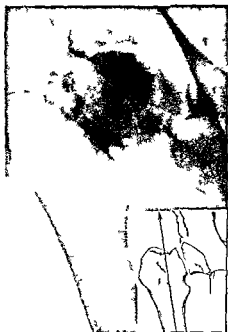


Fig. 6

Two years old adduct on fracture of the neck of the femur treated in a plaster spica Union is still unsound because there is continued shearing stress

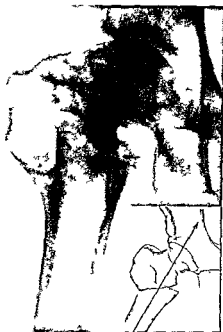


Fig. 71

The shaft of the femur has been abducted by a trochanteric osteotomy. The shearing stress has become an impacting force. On on is now soundly consolidated.

is therefore tempted to discard external splintage altogether or to employ inadequate external splintage or to use external splintage for an inadequate period. But complete and prolonged external splintage is indispensable because the fixation secured by the plate is usually not sustained. Bone reacts to abnormal pressure by absorption and the more tightly the screws have been driven in the more likely it is that they will loosen. If external splintage is inadequate the pressure is increased and every screw subjected to strain becomes loose. Furthermore the screws may loosen by toxic reaction in the bone or by chemical ionisation². Within a few weeks

¹ C. R. Murray: Primary Operative Fixation in Fractures of Long Bones in Adults. *Am. J. Orth. Surg.* 1911; 1: 3-39.

1941 1 3 39
 I n bl and bluck believe I that the loosen up of crews was d e to electrolysis owing to the use of alloys of
 freely ionisable e n nts or p ltes of un ntral and excess of anode. Th ad ceate I v n an alloy
 of neta a wit m n al d r p ltes of n e ntr I potent al (V enafite St ch an l eal
 9) and an y other alloy of n e ntr r e e t w o k o w s th e r t a l e t a l and I as n i k e l c o p p e r a d a
 n e n t e l s o l t e a l and t o x i c and c a n t o n e r a l n i n d e p e n d e n t e f e r e n t a l p o t e n t i a l d i f f e r e n c e
 w h e r e a s o t h e r e t a l and a l o y s c h a n t a n t u m (C L Burke *Cannt Med Ass Jm* 194 11 1)
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 u G n C h t 1940 lex 98 191 lxtl 931)

the fracture is largely dependent on the external splintage for its immobilisation, and rotation and shearing strains lead to the usual and inevitable sequel of non union. If plates and screws are employed it is essential to protect them from every strain. Complete immobilisation by full length plaster casts or by splints must be continued until union is sound. It is possible that future experimental work on new devices for the internal fixation of fractures may succeed in achieving the ideal of complete and sustained immobility even without the aid of splints or plaster. But meanwhile it must be recognised that in the past the failure to supplement internal fixation by adequate external immobilisation has often been responsible for non union /

DELAYED UNION DUE TO INFECTION

The belief that infection is a frequent cause of non union of fractures has survived for many years. Even in 1941, two American surgeons reporting non union in 25 per cent of infected fractures displayed no surprise or regret, and clearly believed that failure in one fracture out of four had been inevitable. In the same year a British surgeon declared that immediate guillotine amputation was often justified, on the grounds that the infected fracture would probably fail to unite, sinuses would continue to discharge and finally a useless limb would be amputated after years of suffering. What a memory of former days! The fact is that infection is not a cause of non union, it is a cause only of delayed union. If non union is allowed to occur, it is due not to the infection but to the inadequate immobilisation permitted by reason of the infection. Immobilisation may be inadequate from the beginning because priority is given to treatment of the infection, complete and uninterrupted immobilisation is sacrificed in order to permit regular inspection of the wound, frequent dressings and antiseptic irrigations. Alternatively immobilisation may be discontinued too soon. A time limit is fixed, and if the fracture has not united within that period, it is assumed that it is not going to unite, the fracture is labelled "un united," complete immobilisation is abandoned and a protective appliance or walking splint is fitted.

Importance of prolonged and uninterrupted immobilisation.—An infected fracture is exactly like a simple fracture, except that each stage of repair is prolonged. Bone is destroyed not only by injury but also by infection, bone is decalcified not only by traumatic hyperæmia but also by infective hyperæmia. In the infected fracture a gap may have to be bridged. Moreover simple traumatic hyperæmia subsides within a few days, whereas infective hyperæmia may persist for weeks or months. Not until infection is healed and hyperæmia subsides can recalcification begin and it may be several months before the infected fracture reaches the stage of repair at which the simple fracture arrives within a few weeks. Throughout these months the infected fracture shows the same susceptibility to shearing and rotation strain. Movement tears the granulation tissue, arrests recalcification and creates a plane of cleavage. On the other hand if immobilisation is complete and uninterrupted, and is continued not only until infection is healed but for several months afterwards until losses are made good and the whole area is recalcified, infected fractures unite with the same certainty as fractures without infection.

Closed plaster 'no dressings' technique—The technique by which infection can be controlled and at the same time the fracture completely immobilised is discussed in Chapter X. The principles are (1) drainage without dressing and (2) immobilisation without disturbance. Free incisions are made, soft tissues are widely laid open so that no deep pockets or recesses remain, and the bone is drained. The wound is lightly packed with vaseline gauze. The fracture is then treated exactly as if it was a closed injury by manipulative reduction, immobilisation in splints or plaster, and if necessary by skeletal traction or skeletal transfixion with pins incorporated in the plaster. No dressing, inspection or irrigation of the wound is necessary. If the drainage operation has been correctly performed the wound and the fracture need not be disturbed. The plaster is changed only after three or four weeks and in the later stages at six or eight weekly intervals. Care is taken to allow no strain or movement of the fragments during the time of replastering. Complete, continued and uninterrupted immobilisation is maintained despite severe infection of bone and soft tissues. In the hands of surgeons skilled in this technique, non union has virtually disappeared as a complication of infected fractures.



FIG 72

Six months old compound fracture of femur with delayed union. There is sequestration, discharge from sinuses and active infection.

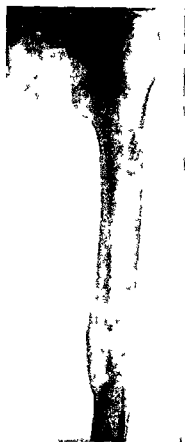


FIG 73

Since infection was active, non union was not established. After simple sequestrectomy and immobilisation for several months the fracture united.

Importance of early sequestrectomy—Throughout the stage of active infection, radiographs show the characteristic features of delayed union. The bone surfaces are decalcified, and the fracture has the appearance of a 'cavity' with a woolly and ill defined margin. It matters not whether the fracture is six, twelve or eighteen months old, it is not an ununited fracture, it is a fracture with delayed union which will unite if immobilisation is continued. The presence of a sequestrum in the region of the infected fracture prolongs the stage of delayed union by causing continued hyperæmia.

and decalcification, and the sooner sequestered fragments are removed the better (Figs 72 73). The persistence of a sinus is evidence in itself of the presence of a sequestrum or foreign body. Even when radiographs fail to disclose a sequestrum the sinus should still be explored, fragments of wood, leather or clothing which are not opaque to X-rays cause the same reaction and the same delay as fragments of dead bone which are opaque. It is often astonishing how rapidly a fracture will begin to unite after many months of indolence when a discharging sinus is healed by removing the sequestrum which was responsible. The operation does not, however, consist in the scraping of sinuses or the scraping of bone. Surgical spoons should be issued only on licence: their use is fraught with danger. Rough scraping may remove some sequestra, but it produces many more by trauma to the



FIG 74



FIG 75

Effect of removing the whole thickness of the shaft at the time of wound excision (Fig 74). The periosteal tube has collapsed and the subperiosteal hematoma is almost obliterated (Fig 75 four months later).

bone and by exposing fresh areas of bone to infection. If possible the sequestrum should be removed by simple dissection with scalpel and forceps, no chisel, gouge or surgical spoon being employed. In these circumstances the wound often heals by first intention, and if the operation is followed by complete and continuous immobilisation the fracture unites soundly within a few months.

Exception to the rule of early sequestrectomy—The one exception to the rule that dead bone should be removed at the first possible moment and as soon as it has separated is the sequestrum which consists of the whole thickness of the shaft of a long bone. Fragments of the shafts of the femur or tibia several inches long may become separated from all soft tissue attachments and undergo avascular necrosis and sequestration. If these large fragments are removed at the time of the original wound excision shortly after injury, or within the first two or three months the surrounding tube of periosteum may collapse and the subperiosteal hematoma be obliterated (Figs 74 75). There is no longer a continuous hematoma between the fragments, and the fracture cannot therefore unite. The position is exactly comparable to that which occurs after diaphysectomy for acute osteomyelitis: an operation now discredited because regeneration of bone so often failed. In such a case it is better to defer sequestrectomy for several months, until a surrounding involucrum of subperiosteal bone has been laid down thereby ensuring continuity of the shaft. When several inches of the shaft of a bone must be reossified the imperative need for uninterrupted immobilisation is very obvious. A single careless movement during the changing of a plaster may refracture the young callus and cause delay and repeated movements or continued strain prevent the fracture from uniting at all.

Non-union of infected fractures—If sequestra have already been removed and the wound is healed, but the fragments have still not been immobilised,



Fig 76



Fig 77

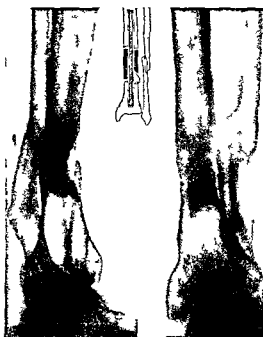


Fig 78

Old infected gap fracture of the tibia with non union due to early removal of a fragment consisting of the whole thickness of the shaft (Fig 77). A bone grafting operation was performed, one long graft being used with two additional short grafts placed end to end between the fragments. After operation infection recurred, there was an acute flare. Stitches were removed and the wound was drained, but the grafts were not removed (Fig 76). Complete immobilisation was maintained by a plaster cast for twelve months. Part of the grafts separated as sequestra and were removed later; most of the graft regenerated, the gap filled and the fracture united (Fig 78).

delayed union passes gradually into non union. The bone ends are even more densely sclerosed, and the intervening scar tissue is even more avascular than in the case of a closed fracture. Immobilisation alone is now valueless. The sclerosis must first be broken down by operation, and a new traumatic hyperæmic decalcification must initiate once more the growth of granulation tissue.

The problems of treatment of the old infected ununited fracture have in the past been dominated by the fear of latent and recurrent infection. It has been suggested that bone grafting should be deferred for twelve months or even for two years after quiescence of the infection. Drilling of the fragments instead of bone grafting has been practised. A preliminary manipulation is sometimes advocated to see whether infection can be lighted up. A two stage operation may be recommended, first freshening the bones and cutting the bed, and two or three weeks later completing the graft if

there has been no flare of infection, presumably if there has been a flare the project is abandoned altogether. When we recall that the source of the non union was not infection but inadequate immobilisation, the situation loses most of its terrors. Recurrence of infection is unfortunate because it may delay recovery by many months, but beyond this little harm has been done. Infective hyperæmia supplements the traumatic hyperæmia of operation, and sclerosed bone is decalcified and scar tissue revascularised with even greater certainty. A successful result may still be expected if immobilisation is prolonged. Precautions should certainly be taken to minimise infection so that the life of the patient or loss of the limb is not endangered. An interval of six months after complete quiescence of infection and healing of the wound will suffice. To wait longer is futile,

because living organisms may remain locked in the dense bone and scar tissue for the rest of the patient's life. However long operation is deferred, the risk cannot be excluded altogether.

If infection does recur pain must be avoided. Drainage is established by removing a number of skin sutures, and the wound is gently packed with vaseline gauze. The graft is not removed (Fig 76). In the same way that removal of sequestra consisting of the whole thickness of the shaft of a long bone should be deferred for several months in order to prevent collapse of the periosteal tube and encourage subperiosteal ossification so removal of the graft in an ununited fracture with recurrent infection should be deferred. The graft assists in maintaining the necessary immobilisation, and it provides a scaffolding for the laying down of granulation tissue and bone. Much of it is replaced by new living bone. After several months loose fragments which are not assisting in fixation of the fracture should be removed in order to prevent continued hyperæmia and delayed recalcification. The



FIG 79

Recurrent infection after bone grafting. The surgeon only immobilised the limb for four months. As might be anticipated the graft absorbed, non union recurred and the operation was wasted.

sinuses at once heal, and after a further period of immobilisation union of the fracture is consolidated. If, on the other hand, imperfect immobilisation is permitted at any stage, the bone grafting operation fails, and the surgeon is faced with an apparently insoluble problem (Fig 79)

NON-UNION DUE TO INTERPOSITION OF SOFT PARTS

The first paragraph of this chapter concludes "there is only one cause of non union of fractures with a continuous hæmatoma between the fragment—the cause of non union is inadequate immobilisation." If there is no continuous hæmatoma and no ossifiable medium between the fragments, union obviously cannot take place no matter how adequately the fracture is immobilised. Diaphysectomy and the removal of large fragments of the shaft of a bone have already been discussed. collapse of the periosteal tube and complete obliteration of the fracture hæmatoma prevent regeneration of bone and bridging of the gap. Similarly, when a small fragment is avulsed from a bone traction of muscles attached to the fragment may cause attenua-



FIG 80



FIG 81



FIG 82



FIG 83

Fractures of the ulnar styloid, patella, internal epicondyle of humerus and tibial tubercle. When a small fragment is avulsed, the fracture hæmatoma may be obliterated by the interposition of soft tissues, and the fracture fails to unite

tion and obliteration of the hæmatoma, as, for example, in avulsion of the epicondyles of the humerus by the flexor or extensor muscles, of the tip of the olecranon by the triceps, the great tuberosity of the humerus by the supraspinatus the tip of a dorsal spinous process by the scapular muscles, of a lumbar transverse process by the erector spinæ, of the anterior iliac spines by the sartorius or rectus femoris, or of the tibial tubercle by the quadriceps.

In other fractures the hæmatoma may be obliterated by an interposed flap of living tissue which seals the fractured surface of one fragment. In fractures of the patella with separation, the surface of the proximal fragment is covered closely by the aponeurosis of the quadriceps expansion. In fractures of the base of the internal malleolus, bony union may be prevented by a flap of periosteum torn from the adjacent surface of the tibia and interposed across the line of fracture. Occasionally in a fracture of the middle third of the shaft of the radius, the surface of one fragment may be sealed in a similar manner by fibres of the pronator teres insertion. In fractures of the shaft of the femur sustained as the result of considerable violence, one fragment may be driven so forcibly through surrounding muscle bellies that, despite traction and manipulation, the fractured surfaces cannot be

brought together, and the interposed mass of living muscle prevents union of the fracture. In all such fractures operative treatment to restore continuity of the bone and apposition of the fractured surfaces is a necessary prelude to the immobilisation by which union is secured.

Summary of the causes of slow union, delayed union and non-union

Slow union with indolence

Impaired blood supply of one or both fragments

Excessive traction, especially when continued for several weeks

Delayed union with cavitation

Inadequate immobilisation, especially rotation and shearing strains

Infection, especially when persistent owing to sequestrat on

Non-union with sclerosis

No continuous hæmatoma—

Diaphysectomy and early removal of large fragments of the shaft

Interposition of soft tissues and avulsion of small bone fragments

All fractures—

Cessation of immobilisation too soon, especially in cases of slow union and delayed union

OPERATIVE TREATMENT OF UN-UNITED FRACTURES

An united fracture with sclerosis of the fractured surfaces may be treated successfully by drilling the sclerosed surfaces and then immobilising the fracture adequately and continuously (Figs 84 85). A bone grafting

operation is still more certain because (a) the preparation of a bed is more effective in cutting through sclerosed bone and scar tissue and initiating hyperemic decalcification and the growth of granulation tissue, and (b) the inlaying of a graft provides an internal splint, augments the necessary fixation and provides a scaffolding for the growth of new bone. These two factors—the removal of sclerosed bone, and increased immobilisation, outweigh in importance any other function or properties of a bone graft. The type of operation to be performed should be determined on these grounds.



FIG 84

Drilling for non union. The traumatic hyperæmia decalcifies the dense bone and initiates a new growth of granulation tissue. If it is followed by perfect immobility the fracture unites.



FIG 85

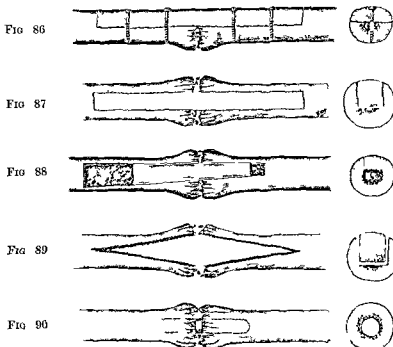
Types of bone graft—There are five types of bone grafting technique (a) only graft (b) simple inlay graft, (c) sliding inlay graft, (d) diamond inlay graft (e) intramedullary peg graft (Figs 86 90).

Onlay graft—About one third of the circumference of the bone is excised from both fragments, leaving a flat surface of raw bone. To this is fixed a massive graft cut from the tibia. Fixation of the graft was formerly achieved by prepared screws of beef bone or ivory,¹ and loosening or rapid absorption of the screws was the main criticism of the technique. It is now customary to fix the graft by vitalium screws which do not loosen or

¹ Henderson. *Jour Bone and Joint Surg* 1934 xx 673

absorb¹ and onlay bone grafting with metallic screw fixation is a most successful operation. Failure is exceptional.

Inlay graft—This is the technique originally described by Albee². The bed is cut with a twin saw. The blades are then separated by the width of two saw cuts so that the graft cut from the tibia is no narrower than the bed. If the technique is accurate the graft is sprung into its bed with some difficulty. When the graft is in position perfect natural fixation is achieved and the added fixation of screws or wire is unnecessary. The inlay operation is applicable to the femur, tibia and other large bones, but difficulties may arise in ununited fractures of the metacarpals, ulna, clavicle and other small bones.



Types of bone graft for non union of fractures—onlay graft with screw fixation, simple inlay graft, sliding inlay graft, diamond inlay graft, intramedullary peg graft.

Sliding bone graft—An alternative type of inlay avoids the necessity for the second operative exposure by which the graft is cut from a healthy bone. A graft is slid down from one fragment across the gap. If the sliding graft is rectangular in shape it is smaller than the bed by the width of two saw cuts and fixation is inadequate. A sliding graft must therefore be wedge shaped and the exact angle of the wedge must be judged with precision.³ Furthermore, since the fragments are often decalcified by disuse the graft may be soft and friable. The technique is therefore reserved for patients who refused to allow the surgeon to tamper with the good leg.

Diamond inlay graft—Gallie⁴ has been an exponent of the diamond shaped bed and graft. The greatest width is at the level of the fracture where there is most sclerosis. This technique is therefore most effective in removing sclerosed bone. It is less effective in maintaining immobility of

¹ Campbell and Boyd: Fixation of Onlay Bone Grafts by Vitallium Screws. *Amer Jour Surg* 1931 II 3 43

² Albee: Bone graft Surgery. New York 1935

³ R. F. Kelly: Application of Wedge Principle in Fashioning a Tibial Bridge Graft. *Bull Jour Surg* 1931 X 23

⁴ (a) *Brit Med J* 1931 II 840

the fragments because it is more difficult to cut a graft coinciding in shape and size with a diamond shaped bed than to cut a graft with a twin saw which exactly fits a simple rectangular shaped bed

Intramedullary peg graft—This is the least satisfactory technique because drilling the medulla to take the peg leaves the greater part of the sclerosed bone undisturbed. Moreover, the medullary cavity itself is plugged so that practically no source of new granulation tissue growth remains. When the bone ends are densely sclerosed the operation is unreliable. Even if sclerosed bone is freely cut away and the wide gap between the fragments is filled with a cricket ball graft pegged into each end according to the technique of Hey Groves¹ the results are still unsatisfactory because the graft is weak where it is dowelled down to a peg and because an intramedullary peg



FIG 91

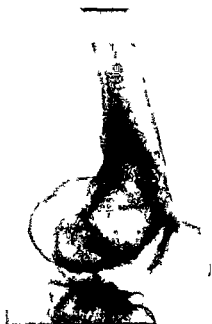


FIG 92

Ten years old ununited supracondylar fracture of femur successfully bone grafted

controls angulatory movement but not rotatory movement so that it fails in its purpose of providing complete internal fixation

Technique of inlay grafting operation—It does not matter how many years the non union has existed. Equally successful results may be secured after ten years as after ten months (Figs 91 92). In older cases there will probably be considerable deformity and shortening of the limb. So far as possible this is corrected before operation. Angulation is corrected by manipulative stretching of contracted tissues and the limb is immobilised in plaster for a week or two until the reaction has subsided. If there is postural deformity of adjacent joints this is similarly corrected. When there is marked shortening skin traction or skeletal traction is maintained for some weeks before operation. The skin is prepared for twenty four to forty eight hours and no touch technique is used throughout.

The limb is exsanguinated and a tourniquet applied. In the upper limb a pneumatic tourniquet must be used to avoid the danger of tourniquet

paralysis. An ordinary sphygmomanometer is effective if it is applied after the limb has been emptied of blood and it is entirely safe if the pressure is maintained at 200 mm. An incision is made straight down to bone without dissection into various planes. The periosteum is raised from the exposed

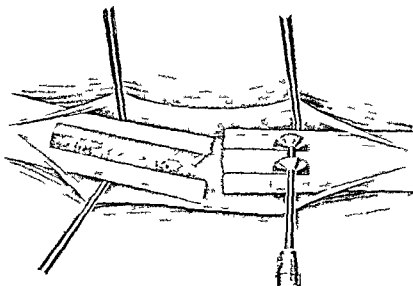


FIG 93

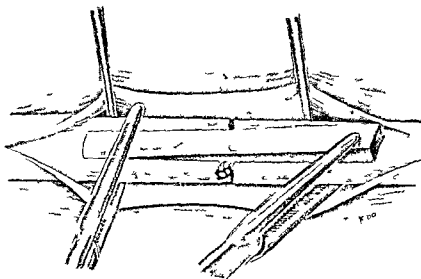


FIG 94

Technique of inlay bone grafting. The bed is cut with a twin saw. The blades are then widened to cut a graft from the tibia which is forced into position. A very accurate fit is essential.

surfaces of the fragments with its muscles still attached so that its blood supply is preserved. All fibrous and scar tissue is removed from the bone ends. Projecting spurs of dense bone which obstruct reduction are removed and the sclerosed bone is drilled or chiselled so that it is broken up. It must not be removed too freely or undue shortening will result. The twin saw blades are then adjusted so that the bed will be

as wide as possible without endangering the strength of the wall of bone remaining on either side. A bed is cut in each fragment (Fig 93). It must be so placed that the apposition, alignment and rotation of the fragments



FIG 90



FIG 92



Intramedullary peg bone grafting. This is not recommended because the graft does not prevent rotatory movement of the fragments and it plugs the medulla.



FIG 94



FIG 96



Diamond inlay grafting of radius and simple inlay grafting of ulna. The greater difficulty in securing an accurate fit of the diamond shaped graft is apparent.

will be accurate when the graft is in position. In fractures of the ulna, radius and clavicle the bed should be about 3 in long, and in the case of the femur, tibia, and humerus 4 in or longer. The saw blades are then separated by a further $\frac{1}{8}$ in. The inside dimension between the saw-
erations must be no less than the width of the bed already cut. The sub

cutaneous surface of the tibia is exposed the periosteum elevated, or fragmented by multiple incisions,¹ and the graft cut exactly to the length of the bed. For the smaller bones the graft must be cut from the middle



FIG 99



FIG 100

Rectangular inlay grafting. When the tibial angulation was corrected there was nearly a 1 in gap between the fragments. It is still unnecessary to divide the fibula and to accept shortening. The gap is easily bridged if fixation is perfect.



FIG 101



FIG 102

Simple inlay grafting. Three previous operations elsewhere had failed because the graft was too small and external fixation was inadequate and was discontinued too soon. Six months immobilisation in a plaster spica was necessary (see Fig 59).

of this surface of the tibia where the bone is about $\frac{1}{2}$ in deep. If a particularly strong graft is required for the femur it is cut nearer to the subcutaneous border where the cortical bone of the subcutaneous and of the

¹ The clinical experience of thousands of bone-grafting operations has proved that it is quite unnecessary to transplant periosteum as well as bone. This has been confirmed experimentally. (G. Loflock and M. S. Henderson. Value of Periosteum in Grafting Operations. *Proc Staff Meet Mayo Clinic* 1910 xv 441).

external surfaces of the tibia is continuous. A graft $\frac{1}{2}$ in or more in thickness may be secured. The graft is at once transferred to the site of fracture and sprung into the bed with the aid of bone holding forceps (Fig 94).

Importance of absolute fixation of the graft—The immediate fixation afforded by the graft must be so perfect that at the conclusion of the operation it is possible to move the limb freely without danger of movement of the fragments or slipping of the graft. If absolute fixation is achieved, it is most exceptional for the operation to fail. On the other hand, if the bed is cut carelessly with an osteotome or chisel, and the graft is merely dropped into it and secured with catgut sutures, there is no immediate fixation, slight rotation and shearing strains tear the columns of cells which are attempt-

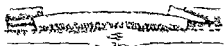


FIG 103

Poor fixation of the graft due to inaccurate cutting, or decalcification of the host bone may be compensated by wedging the graft in its bed

ing to bridge the gap between host bone and graft, and the result is often a failure even when external immobilisation by splints or plaster is as complete as possible. If perfect fixation has not been achieved, either because the technique has been inaccurate or because the bone is decalcified and soft, one of three supplementary measures should be adopted.

1 **Wedging the graft**—The graft may be sprung under the margin of the host bone at one end and fixed at the other end by a wedge of bone driven between it and the host (Fig 103). This measure was first devised for augmenting the fixation of grafts used in arthrodesis of old tuberculous joints, and it is of value when the host bone is very decalcified so that it fails to grip the graft securely.

2 **Combined graft and plate**—In addition to nlaying a graft a vitallium or stainless steel plate and screws may be fixed on the opposite side of the bone (Fig 104). Some surgeons treat every ununited fracture by combined grafting and plating¹. The routine certainly places correct emphasis on the necessity for complete fixation of the fragments but it is not the best method of securing equally complete fixation of the graft.

3 **Fixation of graft with vitallium screws**—This technique is now used almost routinely in onky grafting and the same procedure may be used with an onky graft. Before finally removing the graft from the tibia four holes are drilled, of a dimension equal to the core diameter of the screws to be used, the surface of the drill holes being slightly enlarged with a $\frac{3}{8}$ in drill in order to allow counter sinking of the head of the screw. When the graft is in position the deep layer of the host bone is also drilled, and vitallium screws of the correct length are driven in (Fig 105). It is essential to use screws of a non toxic non electrolytic alloy (see footnote, p 31).



FIG 104

If the fixation of the graft is inadequate it may be supplemented by a vitallium or stainless steel plate on the opposite side of the bone

¹ T. Le Cocq. Ununited Fracture of Both Bones of Forearm. *Northwest Med.* 1940 xxxix, 16.

With a technique sufficiently exact, even wide gaps between the fragments can be bridged successfully. It has been suggested that in ununited fractures of the tibia the united fibula should be divided to allow the tibial fragments to approximate. While it is true that angular deformity of the fibula may need to be corrected before grafting, it should be quite unnecessary to allow approximation of the tibial fragments. There is no necessity to increase the shortening even by $\frac{1}{4}$ in. Several of the cases illustrated in this chapter show successful bridging of gaps (Figs 77, 99 etc). The first essential is to secure absolute fixation of the graft to its bed. The second is to pack the gap with bone fragments including fragments of spongy bone

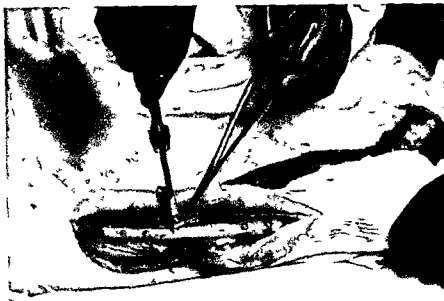


FIG. 100

Fixation of bone graft with vitallium screws. As a rule four screws are sufficient, two in each fragment. In this particular case a double fracture necessitated an 8 in. graft and six screws were used.

from the medullary region which carry with them endosteum, and are the most active source of new cellular growth¹. The third is to protect the graft by adequate and continued post operative immobilisation.

Importance of prolonged immobilisation—As a rule an ununited fracture of the shaft of a long bone unites after bone grafting in three or four months. Nevertheless the same individual variation which is obvious in the rate of healing of recent fractures may occur after grafting for non union. Many operations for non union would have succeeded, but failed because plaster fixation was given up hastily. The passage of time must not be allowed to influence the surgeon. It matters not that six months or perhaps even twelve months have elapsed. Even absorptive change in the graft at the level of fracture does not necessarily mean failure, it is merely an indication that immobilisation is imperfect. The one sign of failure is the

¹ Kilg. Mattas Spongiosa Bone Transplant for Ununited Fractures. *Med Jour of Aust* 1933 1, 506

recurrence of sclerosis of the fractured surfaces after complete disappearance of the intervening graft

Importance of complete immobilisation in plaster—Absolute and un-interrupted immobilisation is essential. The fixation of splints is quite inadequate and may even allow fracture of the graft. Much less severe strain than this can be responsible for traumatic hyperemic decalcification, with absorption of the graft at the level of the fracture, or absorption of the callus between the graft and one of the fragments. Complete plaster casts are essential. If the post operative plaster is padded with wool, a new unpadded cast must be applied in two to three weeks. The following outline gives an indication of the necessary degree of fixation—

Un united fracture	Plaster cast necessary
lower shaft of tibia	from toes to groin (knee flexed),
upper shaft of tibia	full hip spica to the toes
lower shaft of femur	full hip spica
upper shaft of femur	double hip spica
shafts of radius and ulna	from hand to upper arm
shaft of humerus	shoulder spica

The full cast must be retained until union is complete. The practice of changing after eight or ten weeks to a relatively inadequate "protective" cast is as dangerous in the grafted un united fracture as in the recent fracture. This is clearly illustrated in the treatment of un united fractures of the lower shaft of the ulna. In the days when the classical after-treatment was immobilisation for about two months, and then protection by a forearm plaster or leather gauntlet for a further period failure after bone grafting was very common. The graft usually fused with the distal fragment, but the trauma of rotation movement caused hyperemic decalcification of the more vascular proximal fragment, and of the callus between it and the graft. I have records of over fifty un united fractures of the lower shaft of the ulna and in every case a grafting operation has been successful in securing union. The proportion of successful results would however have been very much reduced if immobilisation had not been complete and sometimes very prolonged. One fracture united only after ten months' complete immobility. Several required immobilisation for five or six months. Failure threatened in two cases. In both the complete plaster had been changed to a forearm cast at the tenth week. In one, the hyperemic decalcification absorbed the callus between the graft and the proximal fragment (Figs 106 108). In the other the graft itself absorbed at the level of the fracture. Both ultimately succeeded after complete immobilisation had been reinstituted and continued for several months longer.

Boiled bone, "os purum" and "os novum"—If the main functions of a grafting operation for non union are the removal of sclerosed bone and the fixation of an internal splint, it will be urged that prepared transplants of boiled bone should be equally effective. Un united fractures have in fact been successfully cured by grafting operations in the course of which the graft fell to the floor of the operating theatre was boiled and fixed in position. Specially prepared and shaped grafts may be bought from surgical instrument makers ready to be sterilised and inserted at operation. Animal bone or bone from amputation specimens is freed of

fat and connective tissues and all foreign proteins are extracted. The tissue which is known as os purum is so prepared that a trabecular scaffolding remains capable of easy invasion from the living tissues of the host. There can be no greater proof than the success of boiled bone transplants and transplants of os purum that the main and essential features of a grafting operation for non union are the exposure of vascular tissues and the immobilisation of the fragments by a bone conducting internal splint. But even Svante Orell¹ who advocates the use of os purum, advises as an alternative that a strip of this tissue should be buried under the raised periosteum of the subcutaneous surface of the tibia for



FIG 106



FIG 107



FIG 108

United fracture of radius with un united fracture of ulna which was grafted. Complete immobility was discontinued ten weeks after operation and a short forearm plaster applied. Potation movement caused absorption of callus between the graft and the proximal fragment (Fig 107). When complete immobility was reintituted the graft fused and the fracture united (Fig 108)

two months and then transplanted with the new spongy subperiosteal bone which has surrounded and invaded the strip of dead bone. This partly living bone is described as os novum.

Value of a living autogenous graft.—There can be little doubt that an autogenous graft living at the time of operation is superior to a boiled dead graft. The problem has provoked scientific investigation for nearly a hundred years. The work of Olier was followed by that of Wolff, of Barth, Axhausen and Macewen, and more recently of Hey Groves², Gallie,³ Fell,⁴ Leriche and Polcard.⁵ It is undoubted that the graft never participates in the circulation of the host, and that most of it becomes sequestered and dies. That it is not revascularised is proved by its failure to take up dyes which have an affinity for bone and which when injected intravenously are deposited in all bones accessible from the blood stream. It is also proved by the failure of the graft to participate in the decalcification

¹ Svante Orell. *Surg Gyn Obst* Oct 1934 lix 638. *Jour Bone and Joint Surg* 1937 873

² Hey Groves. *Bone Transplantation* (with good review of earlier literature). *Brit Jour Surg* 1917 v 185

³ Gallie and Robertson. *The Repair of Bone*. *Brit Jour Surg* 1919 vii 211

⁴ Fell. *Biochem Jour* 1909 xxiii 67 and 1930 xxiv 1905. *Jour of Anat* 1917 lxi 2 157

⁵ Leriche and Polcard. *Physiology of Bone*. London 1918

of neighbouring bones¹ for decalcification is an active mobilisation of calcium which can occur only in the presence of a free blood supply. Death of the graft is shown histologically by the absence of cells from the lacunae (Fig. 109). The graft undergoes continued surface erosion. It is invaded by vascular loops of granulation tissue in which new bone is laid down. So complete is this invasion that the original graft is ultimately replaced in its entirety by new bone. The evidence that the main mass of graft is nothing more than a bone conducting medium is overwhelming.



FIG. 109

Low power section of junction of an autogenous bone graft (left) with host bone (right). The lacunae of the graft are empty and the bone is dead but there is proliferation of endosteal and subperiosteal cells and invasion of the graft with replacement by new spongy bone.

But there is also histological evidence in the early stages of multiplication of the surface cells of the endosteum and of the cells beneath the fibrous layer of the periosteum. We know that these cells can be grown and cultured in hanging drop media on a microscope slide. When bathed in nutritive fluids they can grow independently of a circulation of blood. These two layers of an autogenous graft recently cut from living bone do contribute to the cellular growth necessary for repair. The cut surfaces of the bone probably do not contribute so that the objection to cutting a graft with a motor saw—that the heat which is generated may kill the bone cell—is invalid. These cells die in any event and grafts cut with a chisel are no more potent than grafts cut accurately with a motor saw.

¹ See union of fractures considered in further detail in Chapter XXXI together with a review of 834 fractures of the femur and tibia which are analysed from the point of view of rate of union.

CHAPTER III

ADHESIONS AND JOINT STIFFNESS

In the last chapter emphasis has been laid on one fundamental principle of fracture treatment a principle which applies to every type of fracture whether complicated or simple whether infected or not infected whether recent or ununited whether treated by manipulation or by operation—

Every fracture must be treated by complete and continuous immobilisation until union is secured

This complete fixation of the fractured bone involves immobilisation of the joints above and below the fracture. There is a second principle of equal importance which must also be in the mind of the surgeon from the beginning of treatment—

Every joint which does not need to be immobilised must be actively exercised from the first day of injury

Failure to obey the first law of treatment is a source of delayed recovery and may be responsible for non union. Failure to obey the second law is an equally common source of delayed recovery and may be responsible for complications even more serious because they often remain permanently despite treatment.

Stiff fingers due to excessive splintage—If a fracture of the wrist is immobilised in splints which extend over the finger joints (Figs 110 111) the



FIG 110



FIG 111

The bar of the wooden splint and the plaster on the palm prevent flexion and extension of the fingers. This immobility is unnecessary and usually leads to disastrous results.

fingers become so stiff that weeks and months of treatment are necessary to mobilise them once more. They may never recover. Rigidity may remain as a permanent incapacity. The gravity of the disaster cannot be over emphasised. Permanent stiffness of the fingers means permanent crippling. The whole purpose of the limb is lost when the prehensile function of the fingers is lost. Of what use is an upper limb without a hand? If the

patient is a working man he may never work again (Fig 112). But there was no necessity to immobilise the fingers at all. The joints were not stiff at the time of injury; they became stiff in consequence of the treatment. The treatment itself has caused months of delay and even permanent crippling which need never have arisen. The patient would have done better to have stayed at home. Even without any treatment the result could not have been worse. So disastrous has been the result of excessive immobilisation and neglect of joint exercises that Lucas



FIG 11

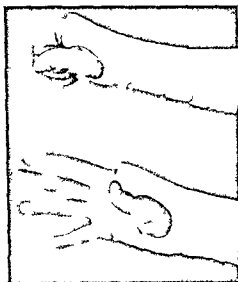
Simple wrist fracture twelve months after treatment as in Fig 111. The patient is trying to make a fist. The hand is so crippled that the man will never work again.

Championniere and others determined to do away with splints altogether. Even delayed union and mal union were not so bad as irrecoverable joint stiffness. The limb was simply supported in a sling and massage and joint exercises were practised daily.

Preventing stiffness despite complete fixation of fracture.—Actually there is no difficulty in combining the two principles of immobilisation of the fracture and mobilisation of the joints. The wrist fracture is completely immobilised in a closely fitting plaster extending from the back of the metacarpal heads to just below the elbow with a single width of strapping or bandage in the palm so that there is no restriction of flexion movement of the fingers. From the first day full bending and straightening movement of every finger joint is practised. The discomfort which may at first be experienced soon passes off and the exercises are continued for not less than five minutes every hour of the day (Figs 113 114). In addition to this the elbow must be fully flexed and fully extended and the limb must be raised to the side of the head and rotated fully in each direction so that *no stiffness can develop in the shoulder*. If the plaster immobilisation is sufficiently complete the patient is encouraged to use the limb for dressing for eating and for light household duties (Figs 116 117). It may even be possible after some weeks for men to go back to work. When the plaster is finally removed every joint of the limb has normal mobility except the wrist itself. Moreover the muscles are well developed wasting has been minimised and disuse decalcification of bones has been prevented. Perhaps the most important point is that the limb has never been disconnected from the brain: the patient has not forgotten how to use it; he is not terrified at the prospect of using it. He has been so busy practising exercises that he has had no time to sit back miserably to lament his fate and to develop functional complications.

The surgeon must first see that no splint plaster strapping or bandage interferes with finger movement. He must not use any position of immobilisation which makes finger exercise impossible. For example the position of right angled flexion of the wrist used in the Cotton Loder

treatment of wrist fractures makes it impossible to flex the fingers. The position is both unnecessary and dangerous (Fig 118). The surgeon's duty is still not done. Not only must there be no obstacle to movement, the



Figs 113 114

Perfect immobilisation of the fracture need not prevent finger movements. The joints are exercised hourly throughout the day.



Fig 115

The shoulder must not be allowed to stiffen. The arm is put over the head and behind the back many times a day.



Fig 116



Fig 117

In many cases household activities and light work can be continued throughout the period of immobility of the fracture.

patient must actually practise the movements which are now possible and he will not do this on his own initiative. His instinct is to secure a sling to preserve absolute immobility and to guard against the slightest movement of any joint. When he has been told to exercise he still doubts or he is afraid or he is unwilling to accept the initial discomforts of finger exercise. Every day of postponement increases the difficulty. The patient must be

seen at least once a day and encouraged, cajoled, stimulated and when necessary bullied into activity. Every joint must be fully flexed, the inter-

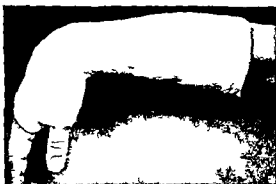


FIG 118

If the wrist is flexed to the right angle it is impossible to bend the fingers. Even though the fingers are not actually splinted, serious stiffness will develop.

phalangeal joints by flexing the fingers tightly into the palm and the metacarpo-phalangeal joints by reaching with the finger tips to the front of the wrist. The index finger must not be allowed to escape attention (Fig. 119). Flexion of this digit may be somewhat difficult owing to the position of the thumb and stiffness confined to this finger is very commonly seen. The patient must be taught to keep the thumb out of the way while he bends the fingers fully.

When the fracture does not involve the wrist but is at a higher level so that immobilisation of the elbow and shoulder joints is necessary, finger exercises are even more imperative. When the plaster is discarded the patient whose hand is normal has an incentive to use the limb and the stiffness of the proximal joints gradually recovers. If the hand is stiff and useless the patient has no incentive. He regards the limb as totally crippled. It never occurs to him to use it. It is then a most difficult problem to persuade him to undertake the exercise necessary for recovery.

Finger exercises prevent stiffness even of immobilised joints—This is not the whole story. Finger exercise is not merely an end in itself. It does much

more than prevent stiffness of the fingers; it actually prevents stiffness of the wrist joint which has been rendered immobile throughout. If two wrist joints with identical injury are immobilised for the same period, one in splints which extend over the fingers and cause functional disuse and one in a dorsal plaster which leaves the fingers free and permits functional activity, although both wrists are equally immobilised, the first becomes very much stiffer than the second. In other words, it is not immobility alone which causes joint stiffness; it is immobility and functional disuse. A fracture of the scaphoid bone with delayed union may require immobilisation for six months or even longer. It might be assumed that this would cause very serious stiffness of the wrist joint. This would be the case if there was complete functional disuse as well as immobility. But where function

involve the wrist but is at a



FIG 119

The position of the thumb is obstructing flexion movement of the index finger. This is a common cause of stiffness of this finger.

is maintained by finger exercises involving contraction and relaxation of the forearm muscles even although the wrist itself is not moved the stiffness is minimised. Immediately the plaster is removed the range of movement is more than half of normal (Figs 10 121) and after only two or three weeks of exercise it is normal.



FIGS 10 121

Movement of the wrist immediately it is removed from plaster after six months immobilisation on a fractured scaphoid. The functional activity of finger exercise has prevented stiffness of the wrist.

Increase in joint movement while immobilised—The possible range of movement of a joint may even increase while it is immobilised in plaster. A fractured wrist taken out of plaster at the third week is very stiff. If it had been left in plaster for two or three weeks longer it would have been less stiff and if immobilisation is needed for several months it continues to gain movement provided only that functional activity is maintained by finger exercise. Old united fractures of the ankle with severe stiffness of the joint are often best treated by immobilisation in a walking plaster. If functional activity is continued the range of movement of the joint gradually increases and it may almost recover to normal during the two or three months that it is immobilised.

CAUSES OF ADHESIONS AND JOINT STIFFNESS

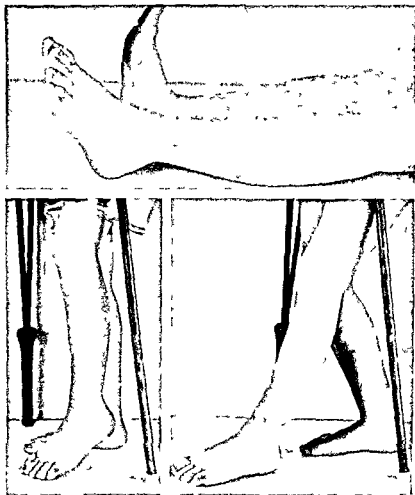
What is the nature of this joint stiffness which is the result not of immobility but of functional inactivity? ¹ It is due to the adhesion of capsular plications, and the glue of which the adhesions are made is the fibrin of sero fibrinous exudates. Any persistent or recurrent sero fibrinous exudation may be responsible. Immobility of a joint cannot in itself cause sero fibrinous exudation, but muscular inactivity is responsible for venous and lymphatic stasis: the circulation is sluggish and the tissues become waterlogged. Here is the source of sero fibrinous fluid which produces capsular and intermuscular adhesions. There may be other sources, and the causes of joint adhesions after injury may be enumerated as follows—

- 1 Functional inactivity and disuse—circulatory and lymphatic stasis and waterlogging of the tissues with sero fibrinous fluid
- 2 Joint injury—traumatic sero fibrinous exudation from torn capsule or from a neighbouring fracture, a source of recurrent exudation if the injured joint is frequently or forcibly moved in the early stages
- 3 Recurrent œdema—reactionary traumatic œdema in the early stages, and recurrent gravitational œdema especially in the lower limbs in the later stages
- 4 Infection near the joint—inflammatory sero fibrinous exudation spreading from a neighbouring focus of infection
- 5 Foreign bodies, especially skeletal traction pins close to joints—reactionary and low grade inflammatory sero fibrinous exudation spreading from the pin track
- 6 Repeated passive stretching and forcible manipulation of a stiffened joint—traumatic sero fibrinous exudation from the stretched and torn adhesions

Functional inactivity and disuse—Figs 120 and 121 show a joint which though immobilised for six months had been functionally active throughout this time and little or no stiffness had developed. Figs 122-124 show the converse. This joint had never been completely immobilised but there was serious functional inactivity and even eighteen months later the joint was still almost completely stiff. The injury was a simple fracture of the malleolus associated with the usual swelling and ecchymosis. The joint was strapped: the patient lay in bed for three weeks and then tried to walk. Meanwhile the foot had dropped. The heel could not be put to the ground. The patient could put no weight on the limb. Weeks went by, functional activity was still impossible. A year later the victim could walk round the kitchen with crutches but no more. The foot and ankle were almost completely stiff. The practitioner labelled it 'arthritis'. The Insurance Company labelled it 'malingering'. Under anaesthesia the adhesions were stretched so that the foot could be raised to the right angle and a plaster was applied in this position. With the painful joints protected and the heel and sole flat on the ground, walking became possible for the first time. Within two weeks the patient walked five miles a day. Within two months the plaster was removed and foot and ankle movement had recovered to

¹ Writ on Jones "Adhesions of Joints and Injury" *Brit Med Jour* May 1936 92.

more than half of normal. In four months recovery was complete. Adhesions round joints which had never been immobilised had been cured by plaster immobilisation with functional activity.



FIGS 122, 123, 124

Eighteen months after simple malleolar fracture of the ankle. Although the joint was never immobilised, functional inactivity has caused adhesion formation. The joint is in so much equinus that the patient cannot walk. The ankle will gain movement while it is immobilised in a walking plaster.

Joint injury—A simple joint injury, not complicated by functional disuse or by other sources of repeated exudation, does not cause serious or lasting stiffness. Even traumatic dislocation of the knee or shoulder joints in which there is extensive capsular injury, causes no more than temporary stiffness. But if the injured joint is treated by early movements and the torn tissues are not protected, there is repeated exudation, and it is the soaking of tissues with fibrinous serum day after day which causes dense adhesions. The practice of treating dislocations and fractures of the shoulder by immediate movements, repeated daily, defeats its own object. Instead of preventing stiffness it aggravates stiffness. The correct treatment is to immobilise the torn tissues until they are healed, to continue exercises of the

distal joints and to begin movements of the injured joint after two or three weeks

Reactionary traumatic œdema—Fractures of the lower forearm are sometimes associated with severe swelling of the fingers. The fingers themselves are uninjured but the œdema spreads from the site of injury especially if the forearm and hand are encased in plaster. The natural instinct of the alarmed patient is to maintain absolute immobility of the fingers. Often the practitioner shares the patient's fear, accepts the swelling as an index of the severity of the injury, and prescribes rest until it has subsided. Exactly the opposite treatment is urgently indicated. (Edema is glue. Adhesions are forming hourly and the more swollen the fingers the more imperative is active exercise. Exercise will not only prevent the adhesions from consolidating but will accelerate the venous return and help the œdema to subside. The fracture must be reduced and immobilised at once so that exercise can be practised painlessly. The patient should be recumbent for a day or two with the limb elevated between pillows in order to reverse the effect of gravity. It is in this type of case

that the most severe and intractable stiffness of the fingers may develop and prompt action is essential.

Gravitational œdema—When a leg or ankle fracture has been immobilised in plaster removal of the rigid external support is followed by œdema of the leg which increases during the day and subsides during the night (Fig 125). This continues until the musculature and circulation of the limb are again toned up by active exercise. If the recurrent œdema is not controlled by elastic support the tissues of the ankle are daily soaked in fibrinous serum and the joint which is already somewhat stiff may become steadily stiffer. This source of adhesion formation can be prevented by applying an elastic dressing as soon as the plaster is removed and keeping it in position for a few weeks until the tendency to œdema subsides.

Rigid clawed toes—When a leg or ankle fracture is immobilised in plaster and toe exercises are neglected the disuse, immobility and recurrent œdema cause stiffness of the toe joints. This is particularly serious if the toes have



FIG 125

Severe œdema of the leg persisting two years after fracture of the tibia and causing serious stiffness of the ankle. This could have been prevented by elastic support applied immediately after removal of the plaster.

been immobilised in the clawed position. The corns which develop on the dorsal surfaces are the least important disability. The significant point is that rigidly hyperextended toes are functionally useless, for they cannot be flexed to the ground (Fig 126). Normally, with every step forward, weight is transmitted to the toes, and as they flex at the metatarsal

phalangeal joints weight bearing beneath the metatarsal heads is reduced. Rigid clawing of the toes interferes with this mechanism, and the patient walks on the metatarsal heads themselves. There is crippling pain and a sensation as of 'walking on small stones'. This source of disability though never described is extremely common and may keep men off their work months after the fracture itself has recovered. It is to be avoided by moulding the plaster to the transverse arch with the central metatarsal heads elevated and all the toes flexed and by insisting on regular toe exercises especially flexion of the metatarsophalangeal joints. Swelling of the toes must be controlled by periodic elevation of the limb.



FIG 116

United fracture of tibia with rigid clawed toes due to disuse and recurrent oedema. Walking on the unprotected metatarsal heads causes severe pain and prolonged disability.

Infection near the joint—A common example of dense adhesion formation round normal joints which though uninjured and not infected are near a source of infection is seen in the septic hand. The infection causes a spreading sero-fibrinous exudation and there is oedema of the whole hand. Very often the infected finger itself is the least serious element of the ultimate disability. There is board-like rigidity of every joint and recovery is then exceptional. Active exercises of every normal finger from the very onset of injury or infection is imperative. The hand must not be totally enveloped in bandage; only the infected finger must be immobilised. The swelling

and oedema must be controlled by elevation of the limb and if there is the least probability that the finger is irreparably damaged it should be amputated at once in order to save the rest of the hand.

Foreign bodies near the joint—**Metallic foreign bodies** may give rise to irritative changes in adjacent tissues with a low grade toxic reaction. If a metal screw, plate or wire has been introduced into a bone so close to the synovial tissues that the reactionary exudate involves the synovium and capsule intractable stiffness arises from the adhesion formation. Fractures of the

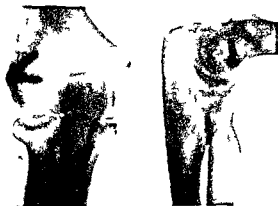


FIG 127

Metallic foreign bodies produce an irritative sero-fibrinous exudation and if close to the synovial reflections of a joint adhesions and limited movement

olecranon may be excellently treated from all other points of view except that wire has been used for fixation, and extension of the elbow remains permanently limited. Fig 127 shows a fracture of the external

condyle of the humerus perfectly reduced by operation but two screws had been introduced close to the joint. When this case was first seen six months after operation there was still only 20° of movement below the right angle. Observation over an interval showed that the recovery of movement had ceased. The screws were removed and within a few weeks movement began to recover once more. Metallic foreign materials should not be used in fractures of the olecranon condyles of the humerus tuberosities of the tibia or patella. Adequate fixation can be secured in all of these injuries with catgut screws nails or wire are superfluous as well as harmful.

Skeletal traction pins—Skeletal traction pins wires and callipers are foreign bodies still more potent in promoting adhesion formation because they penetrate the skin and after some weeks low grade infection of the pin track occurs. Experienced surgeons may be uninfluenced by the case shown in Fig 128 and will maintain that infection of an os calcis pin of such severity as to necessitate guillotine amputation of the leg should not occur. This is true. But slight low grade infection cannot be avoided.¹ There may only be a few granulations a trace of serous discharge or perhaps slight general thickening of the tissues (Fig 129). If the pin track is close to the synovial and capsular reflections of a joint this degree of inflammatory change will be quite enough to bind down these tissues with strong dense adhesions.

Supracondylar skeletal traction—In the supracondylar region of the knee joint a traction pin usually causes adhesions which are far more dense and



FIG 128

A result of skeletal traction

Guillotine amputation following infection of an os calcis pin track

resistant than the simple adhesions of immobility and disuse. Movement beyond the right angle may never recover (Fig 130). Certainly this does not apply to every case—it depends on the degree of reactionary exudation. The bigger the wound at the site of insertion of the pin the greater the danger. The greater the movement of the pin in its track the greater the danger. Ice tong callipers are the worst pins to which the calliper is attached

and which therefore rotate in their track are the next worst Kirschner wires are perhaps the least harmful But no form of supracondylar skeletal traction is entirely safe The method has gained a wholly undeserved popularity and has been responsible for many stiffened knee joints which could have retained normal movement



FIG 129

Diffuse synovial thickening of the left knee from low grade infection of supracondylar pin track The slightest infection in the supracondylar region causes intractable stiffness



FIG 130

Fracture of shaft of femur treated by supracondylar traction three years after injury There is still only 90° of flexion due to adhesions from low grade pin track infection

Tibial tubercle traction—The tibial tubercle is a relatively safe region, for even if slight infection does occur, it is too remote from the periarthicular tissues to cause joint adhesions The objection that tibial traction stretches the knee joint ligaments whereas supracondylar traction does not, applies only when traction is excessive If no excessive traction is used the slight laxity of ligaments which develops after tibial traction is no greater than that which accompanies any form of immobilisation of the knee joint even immobilisation in a plaster spica without traction It recovers fully when the tone of the thigh muscles is regained

Os calcis traction is again unsafe, for the reactionary exudation may bind down the synovial reflections of the subastragaloid joint and cause permanent limitation of inversion and eversion movement Os calcis traction is harmless if the pin is removed as soon as the fracture is reduced and the plaster applied Even continuous os calcis traction may be harmless in fractures of the os calcis where the subastragaloid joint will become stiff in any event If continuous traction is required for a fracture of the leg bones, the pin should be inserted through the lower shaft of the tibia about 2 in. above the ankle joint

Olecranon traction—This is one of the most unsafe regions The elbow joint is particularly susceptible to adhesion formation, and even slight reactionary exudation close to the capsule of the elbow is likely to produce intractable stiffness Fortunately the method is seldom required

Metacarpal head traction—A pin should never be left in the metacarpals for continuous traction The method is entirely unnecessary and there is far too great a danger of stiffness of the metacarpo phalangeal joint

Skeletal traction is not to be condemned out of hand There are occasions when this technique is invaluable But pins must be inserted only with a proper respect for the harm which may result The treatment must be reserved for the cases which really need it The modern tendency to drive

in pins on the slightest provocation is quite unjustifiable. To treat a supracondylar fracture of the humerus or a Pott Dupuytren fracture of the ankle by skeletal traction simply admits a lack of manual dexterity. To treat a simple fracture of the femur by inserting four pins two at the top and two at the bottom is carrying mechanics into surgery to an unwarranted degree. To treat a perfectly straightforward fracture of the humerus by pins at the upper end and at the lower end in order to mobilise the shoulder and elbow from the beginning betrays an imperfect appreciation of the laws of adhesion formation.

Massage and passive stretching—Massage and movements commonly prescribed as a treatment for stiff joints is in fact one of the commonest causes of stiff joints. When the adhesions already formed round a joint are violently stretched or torn there is reactionary exudation which produces fresh adhesions. If this treatment is repeated day after day the traumatic exudation recurs and although every day some adhesions are stretched many more develop. Such treatment delays the recovery of movement in every joint and in the more susceptible joints of the upper limb the finger elbow and shoulder joints measurements show that movement steadily decreases so long as the treatment continues. Enthusiastic masseuses are almost incapable of resisting the temptation to force stiff elbow joints. They should not be tempted. An elbow joint stiff after injury should not be sent to a massage department it must be treated only by the patient's own active exercises. Stiff finger joints are equally susceptible. The one way to convert slight temporary stiffness of a finger into permanent irrecoverable stiffness is to stretch the joint. One often hears of masseuses who soothe the patient's anguish by congratulating him every time the joint cracks. Such treatment is entirely indefensible.

Many other forms of passive stretching are practised. Patients with stiff elbows are encouraged to carry buckets of water or to hang from overhead beams. Increased stiffness is inevitable (Figs 131-133). A surgeon can in fact decide whether an elbow is being passively stretched merely by recording its movement. An angle measurer is used and the range is recorded every other week (Fig 134). Movement recovers by 5° or 10° each fortnight. If movement is not recovering the possibility of passive stretching must be suspected. If movement is less than it was a fortnight previously the surgeon knows that the joint has been stretched.

Manipulation under anaesthesia—It is too generally assumed that if there are adhesions round a joint a manipulation will cure them. Manipulation is a two edged sword. The procedure will undoubtedly break down adhesions but that very process gives rise to a new exudation from the torn fibres which may produce fresh adhesions. The new adhesions cannot be prevented by vigorous and forcible after treatment for as we have seen this produces still further adhesions. Indeed the balance between adhesions cured and adhesions produced may be a very delicate one.

Adhesions must never be stretched under anaesthesia while they are still young and vascular nor while the limb is still subject to recurrent oedema. Manipulation must be deferred until movement is no longer recovering by the patient's own active exercise. This stage must not be determined by mere guesswork. Joints often recover their movement so slowly that the patient himself can judge no difference from week to



FIG 131



FIG 132



FIG 133

The causes of permanent stiffness of the elbow

Passive stretching of any joint defeats its own object and aggravates the stiffness. The elbow is particularly susceptible. Stretching by a masseuse, by carrying weights or by hanging from overhead beams is indefensible.

week The surgeon certainly cannot recall the exact range Accurate measurements are essential If week after week the range is identical, a manipulation may be considered

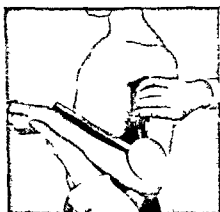


FIG 134

The range of movement must be accurately recorded If it is decreasing the surgeon knows that passive stretching is being employed

by the patient's own active exercise All the benefit will be neutralised if it is followed by repeated passive and forcible stretching Finally, if measurements prove that a manipulation has reduced the movement of a joint the manipulation must not be repeated

If this criterion is insisted upon, it will be found that the indication seldom arises When it is decided to manipulate, the procedure must be carried out with full muscular relaxation and with the utmost caution and gentleness If the joint is manipulated and there is no audible snap of localised adhesions but a gradual giving way of a solid mass of diffuse adhesion the manipulation will do no good and will probably aggravate the stiffness If on the other hand the adhesions are heard to snap and are obviously localised, the prognosis is better provided that too much is not done at one time It is far better to perform two or three gentle manipulations at intervals of not less than six weeks than to perform one severe manipulation which must be followed by a severe reaction After the manipulation treatment must be entirely

STIFFNESS OF JOINTS AND SUDECK'S ACUTE BONE ATROPHY

Adhesion formation near joints is the result of repeated soaking with sero fibrinous exudation and the exudate may be caused by disuse with immobility and oedema repeated injury and passive stretching or infection and foreign body irritation Serious joint stiffening is therefore avoidable Some joints however stiffen more readily than others, the joints of adults stiffen more than the joints of children and there is considerable difference in the susceptibility of different individuals One patient with a fractured wrist has no difficulty in retaining full finger movement whereas the next patient has the utmost difficulty The skin becomes shiny and trophic and unless very frequent active exercises are practised serious stiffness rapidly develops In its most severe form this condition is described as Sudeck's post traumatic acute bone atrophy¹⁶ It usually occurs in the fingers and hand and sometimes in the foot It may complicate trivial as well as severe joint injuries There is pain complete loss of function swelling very marked decalcification of the bones atrophy of the subcutaneous tissues and trophic changes in the skin (Fig 135) It has been

¹ Sudeck "Über die akute posttraumatische Knochenatrophie" *Arch f Klin Chir* 1909 121 147
² Lettich et Fontaine Des Ostéopores Douleuruses Post-traumatiques *Journ med* 1930 xxxviii 61"
³ Middleton and Bruce Post traumatic atrophy of joints *Edin Med Journ* 1934 xli 49
⁴ Fraser (ed) Sudeck's Atrophy *Ann of Surg* March 1934
⁵ Sudeck et al Post traumatic Decalcification of the Foot *Jour Bone and Joint Surg* 1933 xlv 493
⁶ Oppenheimer The Swollen Atrophic Hand *Br J Clin Med* 1934 lvi 416

suggested that the disorder may be neurogenic, possibly due to sympathetic disturbance, and ganglionectomy has been performed

It must be recognised, however, that this syndrome differs only in degree from the ordinary disuse changes which follow every wrist injury when finger exercises are neglected. Once adhesions have begun to form, movement is always painful, functional activity is still further discouraged, and atrophy of the skin, subcutaneous tissues and bones is the usual sequel. The patient who within a few days of injury has swollen stiff fingers, is disinclined to practise exercises who complains of pain and has a tendency to a shiny skin must be watched with particular care. Active exercises must be urged with all the powers of persuasion at the surgeon's command. The possibility of an unduly tight plaster must be excluded. The limb is elevated to relieve œdema. The case must be seen day by day. Exercises are supervised hourly. The temptation to stretch and force the joints must be rigidly avoided. If these steps are taken promptly, the calamity is averted. Only if these steps are not taken does Sudeck's atrophy occur, and I doubt very much whether the condition exists except as the result of neglected disuse changes.



FIG 135

Sudeck's acute atrophy. Almost complete stiffness of fingers and trophic change after wrist sprain. Although the fingers were not actually immobilised the exercises necessary to prevent the complication were never urged, and were not in fact practised.

IMPORTANCE OF QUADRICEPS EXERCISE IN KNEE INJURIES

Muscle exercise during the period of immobilisation is important not only to maintain the circulation and to prevent venous stasis and adhesion formation, but also to preserve muscle control of the joint. This is of particular importance in the weight bearing joints of the lower limb.

Traumatic synovitis of knee—The quadriceps muscle wastes very rapidly after knee injuries. When weight bearing is once more resumed, the joint is imperfectly guarded from the ordinary twists and strains of walking on rough irregular surfaces. The muscle is slow and ineffective, the synovial membrane is therefore nipped and ligaments are strained. Effusion recurs and the patient may be sent back to bed. The muscles waste still more. A vicious circle is established. Swelling of the knee causes wasting of the muscles. Wasting of the muscles causes swelling of the knee. I have known a patient spend two years in and out of bed for no reason except that the thigh muscles were wasted after a simple knee strain. These cases are often cured by bonesetters. Having replaced a non-existent "little bone," they confiscate walking sticks, crutches, and bath chairs and assiduously redevelop the thigh musculature.

Strain of arthritic knee—In other cases an elderly patient with creaking joints twists one knee. The muscle wastes. The joint is unguarded. From then onwards there is pain, swelling, weakness and "giving way." The knee joint has been arthritic for years, but never before has it accounted for symptoms. The opposite knee is equally arthritic but it is painless.

The only treatment necessary is quadriceps exercise to restore the wasted muscle, and within a few months the knee is again free from symptoms

Rupture of knee ligaments—The lateral or cruciate ligaments may be ruptured and if the quadriceps is allowed to waste there is neither ligamentous nor muscle guard of the joint. The incapacity is most serious. A knee cage gives entirely inadequate control and it encourages still further wasting. But if the thigh muscles are perfectly developed even extensive ligamentous rupture is of little significance. Figs 136-138 show a case of complete dislocation of the knee with rupture of the cruciate ligaments. From the first day regular quadriceps contraction was practised for five minutes hourly. Although the joint was completely immobilised in plaster for three months the muscle never wasted. Adhesion formation was minimal and movement rapidly recovered. Despite some laxity of the joint



Figs 136-137

Range of movement and muscular development of the knee at the conclusion of three months plaster immobilisation for complete backward dislocation with rupture of cruciate and lateral ligaments



Fig 138

The quadriceps was never allowed to waste. Exercise was practised hourly every day of the three months. Two months after removal from plaster the knee is functionally normal despite ligamentous instability.

the patient now regards the knee as normal and plays strenuous professional football.

The importance of quadriceps exercise cannot be overemphasised. Whether the injury is to the leg bones, the knee joint, the femur, the hip or the pelvis, wasting must be prevented by regular exercise. No back splint must be applied to the knee without at the same time giving instruction in quadriceps drill. If the muscle is already wasted elastic knee caps, bandages and knee cages must not be prescribed. The best knee cage in the world is but a feeble imitation of the normal thigh muscles. But the muscles can never be redeveloped by electrical treatment three or four times a week. This is quite valueless. The patient must be taught to contract the muscle himself. Even this is not enough, for he imagines that a few minutes exercise each day is sufficient. He must be specifically instructed to exercise every hour of the day for five minutes. An enthusiastic patient who is willing to co-operate can develop a normal muscle in five or six weeks.

CHAPTER IV

MYOSITIS OSSIFICANS AND TRAUMATIC SUBPERIOSTEAL OSSIFICATION

We have seen that passive stretching of joints always defeats its own object and that instead of increasing the range of movement it actually aggravates the stiffness. This is not the only disastrous sequel. It is often responsible for the formation of masses of new bone round the joint. This complication has become known as myositis ossificans traumatica, an unfortunate title because there is no myositis and no ossification in muscles. The condition is quite different from the congenital abnormality, myositis ossificans progressiva, in which bone is actually laid down in the muscle bellies.

TYPES OF PATHOLOGICAL OSSIFICATION

Myositis ossificans progressiva—This disorder is of congenital origin and there is a constant association with congenital shortening of the great toe (Figs 139-141). There are recurrent inflammatory attacks in fibrous



FIG 139

FIG 140

FIG 141

Myositis ossificans progressiva. In this congenital disorder unlike traumatic ossification the bone is found within muscles and its formation is beyond the control of the surgeon.

tissue planes leading to ossification in tendons and in the fibrous intersections of muscles^{1,2}. All the skeletal muscles are ultimately involved, the spine becomes quite rigid and every affected joint is ankylosed. The disease progresses steadily and it is not amenable to any known treatment.

¹ Hutchinson. Multiple Exostoses with Ossification of Tissues. *Arch of Surg* 1896 vii 138.
² Frejka. Myositis ossificans progressiva. *Jour Bone and Joint Surg* 1929 xi 15.

Heterotopic ossification—A second type of pathological bone formation must also be distinguished from traumatic ossification. It occurs in a localised form in tissues remote from the skeleton and from periosteum. The same connective tissues in which pathological calcification is observed are the most common sites of heterotopic ossification. It has been reported in the semilunar cartilages of the knee joint (Fig 142) in abdominal scars and in the tendo Achillis¹. As with myositis ossificans progressiva the bone formation does not arise as the direct result of injury and it is beyond the control of the surgeon.

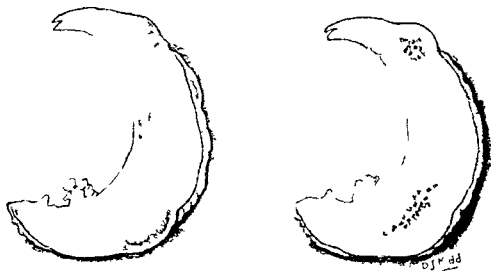


FIG 142

Heterotopic ossification of the semilunar cartilage of the knee joint (from the author's article 'Calcification, Decalcification and Ossification' *Brit Jour Surg* 1934 xx 41)

Myositis ossificans traumatica (traumatic subperiosteal ossification)—The traumatic condition on the other hand is entirely within the control of the surgeon. It is an avoidable complication and its progress can always be arrested². The old theory that osteoblasts escaped from the bone and wandered into muscles laying down bone in their course is erroneous. The new bone formation occurs only within the limits of displaced periosteum and it is nothing more than the ossification of a subperiosteal hæmatoma³. If displacement of the periosteum is prevented and extensive hæmatomata are not allowed to occur the complication is never seen. The true pathology of the condition is most easily recognised at the knee joint.

TRAUMATIC SUBPERIOSTEAL OSSIFICATION

Quadriceps avulsion with ossification—If a young or middle aged patient stumbles so that his knee joint is forcibly flexed by body weight and at the same time he attempts to save himself by strongly contracting the quadriceps the opposing strains cause a fracture of the patella. When a more elderly patient suffers a similar strain the insertion of the quadriceps

¹ Watson-Jones and Roberts 'Calcification, Decalcification and Ossification' *Brit Jour Surg* 1931 xxi, 461
² Watson Jones 'Myositis Ossificans of Quadriceps' *Ind Med Jour* 1933 ii 29 *Canad Med Jour* 1931 xxiv 83

³ Greig 'Surgical Pathology of Bone Injuries' 1931 Traumatic Ossification of Long Bones 10

muscle may give way instead of the bone. The quadriceps is avulsed and it tears the periosteum from the upper border of the patella. If the muscle is stitched back the periosteum is accurately replaced and recovery



FIG 143



FIG 144



FIG 145

Avulsion of quadriceps with traumatic ossification one day four weeks and twelve months after injury. The knee has been immobilised. Bone is formed only in the hematoma within the hood of avulsed periosteum.



FIG 146

Avulsion of quadriceps treated by immediate operative suture. The periosteum has been replaced and the hematoma obliterated. There is no abnormal ossification.



FIG 147

Avulsion of quadriceps treated by early movement and passive stretching. Periosteum has been ruptured and hematoma disseminated. There is scattered bone not fused to the patella.

is complete (Fig 146). If the injury is treated without operative suture by simple immobilisation of the knee on a back splint, the quadriceps remains retracted an inch or more from the raw bone. The interval is filled with blood clot bounded by displaced periosteum. This subperiosteal

hæmatoma undergoes gradual absorption, but at the same time new bone is being formed within it. The two processes go on simultaneously.



FIG 148

Traumatic osteoma from ossification of hæmatoma beneath periosteum avulsed from the femoral shaft

Provided that immobility is continued, the volume of the final mass of bone is considerably smaller than the original hæmatoma, and moreover it becomes firmly attached to the patella (Figs 143 145). Finally, if the injury is treated neither by operative suture nor by immobilisation on a back splint, but the joint is regularly exercised and forcibly flexed, the detached periosteum is ruptured and the subperiosteal hæmatoma is disseminated. Absorption is interfered with and there may even be fresh hæmorrhage, the new bone formed in the hæmatoma is scattered irregularly above the patella (Fig 147). This is the type of bone formation which is described as 'myositis ossificans of the quadriceps'. Clearly, however, the ossification is still within the periosteum, and the question as to whether there will be no bone formation at all, a localised mass attached to the patella, or an extensive formation of scattered bone depends entirely on the treatment.

The same pathology explains traumatic bone formation on the surface of the shaft of the femur, when the origin of the quadriceps muscle is avulsed and bone is formed within the new limits of the periosteum (Fig 148). It explains the bone formation which may follow injury to the ankle, knee, shoulder and elbow joints, and indeed to any joint

where periosteum may be detached by the avulsion of muscles and tendons, or by the tearing away of ligaments and joint capsule.

Traumatic ossification at the ankle joint

—As the result of forcible plantar flexion strains, the anterior capsule of the ankle joint may be torn from the neck of the astragalus. If the foot is immobilised in dorsiflexion the capsule is reattached with minimal new bone formation. If the foot is plantar flexed the periosteum is held away from the astragalus and a bone spur develops which may limit dorsiflexion movement (Fig 149). If the ankle is exercised and passively stretched, a considerable mass of irregular new bone is formed in front of the joint.

Traumatic ossification at the knee joint

—When the internal lateral ligament of the knee is torn in the middle of its fibres, the hæmatoma does not communicate with any bone and repair takes place without ossifica-



FIG 149

Ossification of hæmatoma beneath periosteum avulsed from the astragalus by the anterior capsule of the ankle joint

tion If the ligament is avulsed at its proximal end from the inner femoral condyle, the hæmatoma is subperiosteal and must undergo ossification. The degree of new bone formation, and the localisation or scattering of it, depends on the treatment. It is only the failure to recognise and to understand the pathology of the condition which has led authors to describe this as a special clinical entity with the title of "Pellegrini Stieda's disease".¹³ It differs in no respect from the ossification of any traumatic subperiosteal hæmatoma.

Traumatic ossification at the shoulder—When the clavicle is dislocated upwards at its outer end there is tearing of the acromio clavicular ligaments, and if displacement is severe, avulsion of the conoid and trapezoid ligaments from the coracoid process. If the dislocation is completely reduced and adequately immobilised, the ligaments become reattached. If the dislocation is not reduced the subperiosteal hæmatoma at the site of



FIG 150

Subperiosteal ossification of avulsed conoid and trapezoid ligaments due to unreduced upward and backward dislocation of clavicle



FIG 151

Traumatic ossification round shoulder joint due to passive stretching after dislocation of the joint

avulsion of each of these ligaments undergoes ossification (Fig 150). Dislocations of the shoulder joint itself are seldom left unreduced, so that avulsed capsule and ligaments are usually accurately replaced. But these structures may be detached once more by early forcible movements, and a hæmatoma results which undergoes ossification (Fig 151).

Traumatic ossification at the elbow—It is in the elbow joints of children that the complication is most commonly seen. This is simply because periosteum is more easily stripped in the child than in the adult, and because in children the elbow joint is dislocated more commonly than any other joint. Obviously the condition will be more frequent after dislocations than after fractures, because muscle and capsule insertions into periosteum must necessarily be avulsed in a dislocation. From what has been said of other joints it will be clear that the two causes of subperiosteal hæmatoma ossification after elbow dislocation are—

- (1) failure to reduce the dislocation promptly, the longer the bones are displaced the longer the periosteum is displaced, and the more advanced is the ossification of the hæmatoma, and

¹ Odessky. The Koehler-Pellegrini Stieda Syndrome. *Lyon Chir.* 1937 xxxiv 272.

² H. S. Callen. Pellegrini Stieda's Disease. Manifestation in knee of lost traumatic clanges common to other joints. *Radiology* 1937 xxix 158.

³ H. L. Brumbaugh. Calcifying Tendinitis Traumatica. *Amer Jour Surg* 1940 xlviii 621.

(2) passive stretching and forcible vigorous movements before the periosteum is firmly reattached to the bone

It must be emphasised that it is *passive* stretching and not active exercise which redisplaces the periosteum. It is the deliberate stretching of the stiff joint by an over enthusiastic masseuse or anxious parent, or by the drag of heavy buckets of water or bags of sand, which is responsible. The joint has usually been immobilised in flexion, and it is extension movement which is limited and which may be forced. The subperiosteal ossification is therefore seen in front of the joint, in some cases due to avulsion of the brachialis anticus from the ulna, in some due to avulsion of the forearm muscles from the condyles, and in some due to avulsion of both (Figs 152 153). When both have been avulsed, subsequent tearing of the periosteal walls of the hæmatomata may allow them to communicate, and the joint then becomes ankylosed by a continuous bridge of bone.



FIG 152



FIG 153

Traumatic ossification due to passive stretching after dislocation of elbow joint. In Fig 152 the brachialis anticus is avulsed from the ulna. In Fig 153 the common flexor origin is also avulsed from the humerus.

Treatment of traumatic ossification at the elbow—The first sign of the complication is the radiographic evidence of a cloudy shadow in these situations (Fig 154). The shadow gradually becomes more dense and consolidated but since the hæmatoma is continually absorbing, the final bony mass is much smaller than the original shadow. The one essential treatment is to stop the passive joint stretching which is responsible. There must be no massage or any other form of treatment by a masseuse. The child must not be permitted to carry heavy weights, or to hang from overhead beams by the affected limb. Movement must be allowed to recover at its own rate by the patient's own guarded activity.

The absolute immobility sometimes recommended is neither necessary nor advisable. Tearing away of the periosteum and hæmatoma formation is the result of passive stretching not of active exercise. The only treatment necessary is to prohibit passive stretching. Recovery is no more rapid if active exercise is also prohibited by completely immobilising the joint in

plaster. On the other hand this complete immobility allows consolidation of the adhesions which have also occurred from the passive stretching and permanent stiffness and limitation of extension are then inevitable. If the patient's own activity is permitted the hæmatoma will still absorb the new bone will still shrink and yet mobility will gradually improve (Figs 154 155).

Operative removal of bone spurs and bridges which are locking the joint must not be undertaken in the early stages. An operation performed before the ossifying hæmatoma is absorbed will only disseminate it and add to the ossifying area the new hæmatoma of operation. There is no indication for operation until the bone is finally consolidated and until spontaneous recovery of movement is shown by actual measurement to have ceased. If a bone spur is definitely obstructing movement it may then be removed but very often the stiffness is due to the adhesions which were also caused by the stretching rather than to the bone block and the range of movement may not be improved by operation.



FIG 154

Subper osteal ossificat ion due to pass ve stretching after supracondylar fracture. Prolonged immobilisation would cause permanent stiffness because the joint has been seriously traumatised.



FIG 155

Same case as Fig 154 treated thout immobilisation. Passive stretching was prohibited but active exercise encouraged. The bone has consolidated just as rapidly as if the joint had been immobilised.

Rare type of traumatic ossification at the elbow—A periosteal hæmatoma may form at the elbow joint from disinsertion of the biceps tendon even when there has been no dislocation or fracture¹. The periosteum of the radius is stripped and retracted several inches by the elastic recoil of the muscle. If it is not reattached an extensive subperiosteal hæmatoma remains which undergoes ossification. In one case this injury was sustained by a man who shook hands with a horse by flexing his elbow and placing his supinated forearm below the animal's raised forelimb. At the moment that the biceps was tense the horse decided to shake no more put his foot to the ground and disinserted the muscle. The retracted tendon was exposed through a 1 in incision in the arm and through an incision over the back of the interosseous space long forceps were introduced by which the tendon could be pulled down and sutured to bone. In this way the hæmatoma was obliterated without an extensive dissection in front of the joint. Recovery was complete and there was no trace of the bone formation usually seen after this injury.

S. Nèque and B. Rite. Ruptures and Disinsertions of the Tendon of Biceps. (Report one case abstract of literature). *Jour. de Chir.* 1935 x 1 34.

CHAPTER V

AVASCULAR BONE NECROSIS

Surprisingly little attention has been paid in fracture treatment to the problems of avascular necrosis of bone and articular cartilage. Every group of fractures may be influenced by this complication. Treatment may be entirely changed. Incapacity periods may be extended from months to years. Permanent disability may take the place of complete recovery. It is of the most vital importance.



FIG. 166

Sequestration of upper femoral epiphysis in acute septic arthritis of the hip joint. The epiphysis has not participated in neighbouring decalcification and it is therefore avascular.

the radiographic appearances are unchanged. Shortly, however, neighbouring bone reacts with an active hyperemia which is manifested in osteoporosis of the living bone. This osteoporosis can occur only in the

Pathology of avascular, aseptic necrosis—It was König in 1888 who described the pathology of osteochondritis dissecans as "quiet necrosis". This was probably the first recognition of what is now known as aseptic or avascular necrosis. The blood supply of the bone may be lost through embolism or thrombosis of its vessels^{1,2} or it may be cut off by a fracture^{3,4}. The pathological changes which supervene are to be described in three stages.

Onset of necrosis—There is immediate cellular death of the avascular tissues. The marrow elements change to a formless oily debris, bone cells disintegrate, and lacunae become empty tombs. When a joint surface is involved patches of necrosis appear, separated by areas where articular cartilage has survived by direct nutrition from the synovial fluid. The general architecture of the bone remains undisturbed so that

¹ Iliometer. Repair of Bone in the Presence of Aseptic Necrosis from Fractures Transplantations and Vascular Obstructions. *Jour. Bone and Joint Surg.* 1930 xii, 709.
² R. L. Coley and I. M. Moore. Calcium Disease in Bones and Joints. *Ann. Surg.* 1910 cxi, 1065.
³ Watson Jones and Roberts. Calcification and Ossification. *Brit. Jour. Surg.* 1914 xxi, 461.
⁴ Watson Jones and Roberts. Pathological Calcification and Ossification. *Proc. Roy. Soc. Med.* 1933 26 (B) 253.
⁵ Watson Jones and Roberts. *Brit. Jour. Radiol.* 1931 vi, 851.

presence of a free blood supply, for the calcium is carried away by the blood stream. Avascular bone cannot be decalcified—it retains its original calcium content. It is for this reason that a sequestrum can be distinguished radiographically in infections of bone (Fig 156) and whether the blood supply is cut off by infection or by injury the same differentiation between dead and living bone becomes possible. The necrotic area preserves its original density and it may even appear by contrast to be increased in density.

Stage of regeneration—Active hyperæmia of the neighbouring bone initiates a growth of granulation tissue. Capillary loops and a fibrous stroma invade the necrotic area. Phagocytes both multinuclear and mononuclear resorb the dead marrow and grow along the Haversian canals which become enlarged to several times their original size. They are followed by bone forming cells and bone resorption and bone formation go on almost simultaneously. The Haversian canals are rebuilt layer upon layer and they gradually resume their normal proportions. By this creeping substitution both form and architecture are preserved. The process is identical with that by which a bone graft cut off from its circulation is invaded and replaced by living bone (Fig 109 p 48). The replacement may be traced radiographically by the decalcification which accompanies revascularisation. In the earlier stages the appearances often suggest fragmentation of the dead bone because tongue like inroads of vascular decalcified granulation tissue surround the islets of avascular dead bone. Similarly localised areas of decalcification give an appearance of cyst formation.

Stage of healing—Regeneration may occupy many months or even years. The newly formed bone is soft and easily distorted but ultimately it regains the full strength of original bone. Articular cartilage on the other hand suffers more permanent damage. It is largely replaced by fibrous tissue and fibro cartilage. Early weight bearing encourages collapse of the subchondral bone and it may be responsible for irregularity of the joint contours. Even if there is freedom from weight bearing the new fibro cartilage is so imperfect that degenerative arthritis often develops.

Clinical applications of avascular necrosis—In every fracture minute detached fragments of bone are deprived of their blood supply. Indeed over the whole fractured surface bone cells die, lacunæ are empty, and there is a thin film of avascular necrotic bone. This is invaded and replaced by living bone and it represents the normal process of repair (see Fig 1). In the treatment of non union of a fracture the graft is avascular necrotic bone which is gradually substituted by living bone. Similarly when a recent fracture is reduced by open operation considerable areas of bone may be stripped free of periosteal and vascular attachments. Even the whole thickness of the shaft of a long bone may be rendered avascular, become necrotic and undergo creeping substitution. In short complete loss of the blood supply of one fragment is no barrier to union. Repair is delayed and it is sometimes complete only after twelve months, two years or even longer. Nevertheless if immobility is sustained for an adequate period sound union of the fracture will be achieved.

If the avascular fragment carries with it the articular cartilage of a joint the outlook is wholly changed. Repair will still be slow, but more than this it will now be imperfect. Replacement of the hyaline cartilage with fibrous



Fig. 157



Fig. 158



Fig. 159

Fracture neck of femur. At the time of injury both fragments are equally calcified (Fig. 157). Seven weeks later there is diffuse decalcification of all bones except the femoral head which is therefore avascular (Fig. 158). Ten weeks later the evidence has disappeared for there is no longer decalcification of the other bones (Fig. 159). An accurate prognosis is possible only if radiographs are taken during the second month.

tissue or with an imperfect fibro cartilage is almost inevitable. As a rule the joint space becomes progressively narrowed movement is seriously restricted and degenerative arthritis supervenes.

Early radiographic diagnosis of avascular necrosis—The early radiographic diagnosis is based upon the apparent density of avascular bone¹. Since the bone has no blood supply its calcium content cannot change appreciably whereas neighbouring vascular bone can undergo disuse decalcification. In these circumstances avascular bone appears dense by contrast. It must be recognised however that the density is purely relative that it is not a true hypercalcification and that the evidence is not available unless neighbouring bone shows decalcification. The diagnosis cannot be made during the first few weeks after injury before disuse decalcification has begun. Moreover it cannot be made in the later stages after the resumption of functional activity. The whole tendency of modern fracture treatment is to minimise disuse change by early functional activity and the sign of relative density is transient (Figs 157-159). It is true that after several months other radiographic signs of avascular necrosis may appear particularly in the lower limb where the fragility of necrotic bone is shown by a tendency to disintegration and crushing under the pressure of weight bearing. But the diagnosis should be made before this stage so that the harmful effect of early weight bearing can be avoided. Moreover bone disintegration and degenerative arthritis do not always develop so quickly there is sometimes an interval of several years before the onset of these sequelae and if the passing shadow of relative density has been overlooked a completely erroneous prognosis may have been given. It is important therefore in the case of all fractures sustained in regions where avascular necrosis may supervene to take radiographs during the second third and fourth months at about three weekly intervals. If the shadow of relative density appears in any one of these films the warning must not be ignored.

AVASCULAR NECROSIS OF THE HEAD OF THE FEMUR

Blood supply of the femoral head—The nutrient and periosteal vessels of the femoral shaft extend as high as the trochanteric region and lower part of the neck but they do not contribute to the blood supply of the head of the femur. The femoral head is supplied from two sources—(1) capsular vessels (2) ligamentum teres vessels. The vessels of the capsule enter the bone through foramina at the site of capsular attachment completing their course within the bone or they run on the surface of the neck in retinacula reflected from the deep aspect of the capsule finally entering the bone in the subcapital region. These vessels supply the upper part of the neck of the femur and the greater part of the head. The ligamentum teres vessels are well defined in children and usually remain patent throughout adult life^{2,4} they supply an area of bone and articular cartilage in the region of the fovea centralis. There is no anastomosis between these two sets of end arteries and if either group of vessels is obliterated by injury thrombosis

Watson Jones and Roberts. Significance of the Density of Bone Below. *Br. Jour. Surg.* 1934 xxi 46.
 * Chondro and Kreisler. Blood Supply of Ligamentum Teres. *Jour. Bone and Joint Surg.* 1937 xxx 784.
 † Wolcott. Circulation of Head and Neck of Femur. *Jour. Amer. Med. Assoc.* 1933 ci.
 ‡ Kilbinger and Friedlander. Vascularity of Ligamentum Teres. *Bull. Hosp. Joint Diseases* 1940 July.

or embolism the corresponding part of the head of the femur loses its blood supply.¹ The capsular vessels may be interrupted in the capsule itself as the result of traumatic dislocation or by surgical exposure of the joint or they may be damaged after entering the bone by fractures in the upper part of the neck. The vessels of the ligamentum teres may be torn by the injury of traumatic dislocation of the hip or they may be damaged by forcible rotation movements or manipulation of the joint causing stretching of the ligament over the margin of the acetabular fossa.

Fracture of the neck of the femur²—*Basal fractures* in the trochanteric region of the femur lie below the site of entry of capsular vessels and do not interfere with the blood supply of either fragment. *Transcervical fractures* occur in a dangerous region. Capsular retinaculæ usually remain attached to the proximal fragment but the blood supply they carry is precarious. If for example the fracture is not promptly immobilised by

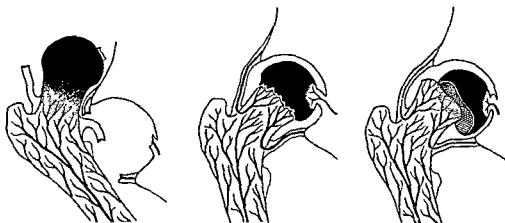


FIG 160

The femoral head is supplied with blood by vessels from the capsule. If these are damaged by a traumatic dislocation, a high fracture of the neck, or epiphyseal separation, avascular necrosis supervenes.

ruining the fragments, unguarded movement may damage the remaining vessels. Forcible manipulation during reduction of the fracture may be equally dangerous. Fractures at this level are more liable to interfere with the blood supply of the head in children than in adults. Even low fractures near the basal region, which are seldom complicated in adults, sometimes cause avascular necrosis in children.³ *Subcapital fractures*—A high subcapital fracture may lie above every capsular attachment to the head, no matter what precautions are taken, the blood supply is then lost to all parts of the head except in the region of the fovea (Fig 161). Patients who sustain this injury are usually old, and the vessels of the ligamentum teres may already have been inadequate, so that quite frequently the whole femoral head including the region of the fovea shows evidence of avascularity (Fig 162). *Operative exposure of femoral neck fractures*—Open exposure of any fracture of the neck of the femur with division and retraction of the capsule

Watson J. Avascular Necrosis of the Femoral Head. *J. Bone Joint Surg. Br.* 1951; 33: 100. (Cited in Watson J. Avascular Necrosis of the Femoral Head. *J. Bone Joint Surg. Br.* 1951; 33: 100.)
 1. Watson J. Avascular Necrosis of the Femoral Head. *J. Bone Joint Surg. Br.* 1951; 33: 100. (Cited in Watson J. Avascular Necrosis of the Femoral Head. *J. Bone Joint Surg. Br.* 1951; 33: 100.)
 2. Watson J. Avascular Necrosis of the Femoral Head. *J. Bone Joint Surg. Br.* 1951; 33: 100. (Cited in Watson J. Avascular Necrosis of the Femoral Head. *J. Bone Joint Surg. Br.* 1951; 33: 100.)
 3. Watson J. Avascular Necrosis of the Femoral Head. *J. Bone Joint Surg. Br.* 1951; 33: 100. (Cited in Watson J. Avascular Necrosis of the Femoral Head. *J. Bone Joint Surg. Br.* 1951; 33: 100.)



FIG 161

The part of the femoral head supplied by capsular vessels is relatively dense and therefore avascular. The part supplied by the ligamentum teres is decalcified and therefore vascular.

vessels the more grave is the complication. Furthermore the capsule of the joint is also torn and according to the degree of violence this source of blood supply is either impaired or completely lost. Avascular necrosis of the whole head with degenerative arthritis and sometimes with complete disintegration of the bone occurs in over 30 per cent of traumatic hip dislocations. The incidence is increased if further injury to surviving capsular vessels is inflicted by unduly forcible manipulation or early passive movement. If operative reduction with extensive stripping and division of the capsule is needed for a late unreduced dislocation, avascular necrosis occurs in nearly every case.

Epiphyseal coxa vara—Separation and backward displacement of the upper femoral epiphysis may result from a single acute injury but more often it develops gradually and apparently spontaneously in abnormally heavy children who suffer from pituitary dystrophies. As the epiphysis rotates backwards on the neck its

endangers the blood supply of the head. Vessels which escaped damage by the bone injury on which the femoral head depends for its survival may sustain injury in the capsular part of their course. For this reason operative exposure has now been abandoned and nailing of the fracture is performed by an extra-articular technique through an incision over the trochanter with radiographic control. The incidence of avascular necrosis in nailed fractures of the femoral neck has been reduced thereby from over 30 per cent to 15 per cent.

Dislocation of the hip joint—Traumatic dislocation of the hip joint is necessarily accompanied by rupture of the ligamentum teres and loss of blood supply to part of the head of the femur in the region of the foramen. Even if the area is limited the joint must suffer some degree of damage and complete recovery with a normal hip joint is exceptional. The more extensive the area supplied by ligamentum teres



FIG 162

The whole femoral head is dense. Capsular vessels have been interrupted by the fracture and the ligamentum teres vessels are inadequate. Inset—A Lorenz osteotomy is better than nailing if the femoral neck is avascular.

central position in the acetabulum is maintained by an increasing degree of external rotation deformity of the whole limb. In former days it was customary to attempt reduction of this deformity by forcible correction under anaesthesia. If the limb is strongly rotated inwards and correction does not take place between the femoral neck and epiphysis, the epiphysis is twisted into the back of the joint, the ligamentum teres is stretched over the margin of the acetabular fossa and the vessels within the ligament are injured. If the position is maintained for several weeks or months by a plaster spica, destruction of the ligamentum teres vessels is almost inevitable. This mistake was often made in the past because if antero-posterior radiographs are taken with the limb in strong internal rotation a radiographic illusion gives the appearance of accurate reduction even when the epiphysis is still grossly displaced (see Chapter XXIX). Avascular necrosis supervened with degeneration of the articular cartilage, narrowing of the joint space, stiffness of the hip and



Fig 163



Fig 164

Low trans-cervical fracture in a child causing avascular necrosis

arthritic change.¹ This complication can usually be avoided if displacement of the upper femoral epiphysis is corrected not by forcible manipulation under anaesthesia but only by cautious and gradual traction.

Legg-Perthes' disease of the hip (pseudo coxalgia)—When fracture of the neck of the femur or dislocation of the hip joint occurs in young children the complication of avascular necrosis is manifested by relative density of the epiphysis, followed after several months when revascularisation is taking place by an appearance of fragmentation, and if weight bearing is permitted by flattening of the contour of the head (Figs 163 164). These epiphyseal changes attributable to rupture of the ligamentum teres or interruption of the capsular blood vessels, are strikingly similar to the changes of Legg-Perthes' disease of the hip joint. So strong is the clinical resemblance that it must be assumed that Legg-Perthes' disease is a simple avascular necrosis due to concealed injury or thrombosis of the vessels of the ligamentum teres or capsule.²

Congenital dislocation of the hip joint—In former days, congenital

¹ Waldenström. Necrosis of Femoral Epiphysis owing to Insufficient Nutrition through Ligamentum Teres in Epiphyseolysis. *Acta Chir Scand* 1914 lxxv 145

² Jackson Burrows points out that although coxalgia (Legg-Perthes' disease) is proved experimentally to be a condition of necrosis of bone followed by repair, the necrosis differs from ordinary aseptic necrosis in allowing lysis of bone trabeculae. He believes that the lysis may be due to venous obstruction rather than articular anastomosis. Coxalgia. Its Pathology and Kinship. *Brit Jour Surg* 1911 xxix 21

dislocation of the hip joint was reduced by forcible methods. The violence of the manipulations was easily capable of damaging the vessels of the ligamentum teres and this explains the frequency with which typical Legg Perthes disease supervened. Operative exposure of congenital dislocations was complicated in a similar way and the operation of reconstructing the upper lip of the acetabulum by the insertion of a bone graft fell into disrepute for this reason. In these cases it was the vessels of the capsule rather than the vessels of the ligamentum teres which were damaged but the sequel was the same. Orthopaedic surgeons now reduce congenital dislocations with great gentleness and care and the complication of Legg Perthes disease is seldom seen. Moreover in a series of over thirty congenital dislocations I have bone grafted new acetabular margins taking care never to divide the capsule or even to expose it and only in one case has there been minor epiphyseal disturbance.

Monarticular osteoarthritis of the hip joint—In the same way that a vulnerable blood supply explains the frequency of Perthes disease of the hip joint in children I believe that it explains the frequency of osteoarthritis of the hip joint in adults. Osteoarthritis occurs far more commonly in this than in any other joint. The radiographic appearances are similar to those of degenerative arthritis due to avascular necrosis after dislocation of the joint: there is the same mottling, irregular density, appearance of cyst formation, narrowing of the joint space and flattening of the contour of the head. When exposed at operation areas of avascular necrotic bone and articular cartilage are evident and operative experience suggests that the vessels of the ligamentum teres are seldom patent in osteoarthritic hips. Is it not highly probable that the usual cause of osteoarthritis of the hip joint is avascular necrosis and that the source of deprivation of the blood supply may be obvious as in the case of fractures and dislocations or it may be concealed and due to minor strains and twists which injure the ligamentum teres and cause thrombosis of its vessels?

Summary of Avascular Necrosis of the Head of the Femur

1 Blood vessels of the capsule of the hip joint and of the ligamentum teres are each of importance in maintaining the nutrition of the femoral head.

2 If either of these groups of blood vessels are destroyed avascular necrosis of the femoral head supervenes causing degenerative arthritis in the adult and epiphyseal disturbance in the child.

3 Capsular vessels may be destroyed by injury to the capsule in traumatic dislocation of the joint, violent manipulation of the hip or the surgical exposure of fractures, traumatic dislocations or congenital dislocations of the joint.

4 Capsular vessels may be destroyed in their course through the neck of the femur by subcapital fractures in adults and transcervical fractures in children.

5 Ligamentum teres vessels may be destroyed by rupture of the ligament in traumatic dislocation of the hip or by stretching the ligament by forcible internal rotation in epiphyseal *colla vara* or congenital dislocations.

6 Legg Perthes disease in children and osteoarthritis of the hip in adults are manifestations of avascular necrosis. The deprivation of blood supply may be due to the obvious causes of fracture or dislocation or to the concealed causes of minor injury or inflammatory thrombosis.

Fracture of the Neck of the Femur

Sir Astley Cooper wrote of fracture of the neck of the femur in 1823

It is impossible even for a few hours to preserve exact apposition of the fragments and the surgeon should not be held responsible for a fracture over which he has so little control. Control by the surgeon was achieved with the introduction of the three flanged steel nail by which exact apposition and complete immobility of the fragments could be maintained even when early weight bearing was permitted. A perfect result depended only upon the surgeon's skill in accurate insertion of the nail. The recognition that impairment of blood supply might delay union for twelve months or two years and that complete immobility of the fragments was needed throughout this time made it still more obvious that nail fixation was the treatment of choice. But we now know that if impairment of blood supply of the femoral head is complete and avascular necrosis supervenes a steel nail ploughs through the fragile bone, redisplacement occurs, the bone disintegrates and degenerative arthritis supervenes. In these cases the nailing operation is no longer a brilliant success. It is better to adopt the second best plan of treatment and perform an osteotomy of the Lorenz type in which an arthroplasty is established between femur and pelvis, the necrotic head of the femur remaining sequestered and immobile in the acetabulum (Fig 162 inset). What is the evidence on which the diagnosis of avascular necrosis may be established? In which circumstances should the surgeon change his plan of treatment from the nailing operation to the osteotomy?

1 Relative density of the femoral head—If the fracture is several weeks or months old, limited function has already caused general disuse decalcification and if the femoral head is relatively dense, impairment of its blood supply is proved. In these circumstances the nailing operation should not be employed. On the other hand, in fractures a few days old the diagnosis cannot be made. An accurate diagnosis could of course be established by deferring operation for several weeks and noting whether relative density of the head appeared, but since delay in immobilising the fragments may itself precipitate the complication by causing further damage to surviving vessels, this course is not justifiable. The nailing operation should be performed without delay, radiographs being taken at three weekly intervals. If relative density is shown even if only in one film, the surgeon must be warned. It does not necessarily follow that complete necrosis and rapid disintegration is inevitable (Figs 165-168). It is true that degenerative arthritis will probably supervene in the course of time, but meanwhile many years of good function may be enjoyed. Two precautions must be taken. (1) Union will be slow. It will be complete only in twelve months, two years or even longer. Throughout the greater part of this time stability of the bone will depend largely upon the nail and it should not be removed even twelve or eighteen months after operation because immediate refracture will almost certainly occur. (2) Weight bearing should be deferred for many months in order to prevent crushing of the fragile bone and damage to the devitalised articular cartilage. Immobilisation of the joint is not advisable, revascularisation is best promoted by active non weight bearing exercise.

2 Increasing penetration of the nail and shortening of the neck—Comparison of films taken at two monthly intervals may show progressive shortening of the neck and increasing penetration of the nail (Figs 169 171) The most reliable measurement is from the point of the nail to the articular surface Increasing penetration indicates abnormal fragility of bone and is evidence of necrosis It is most pronounced when early weight bearing has been permitted Ultimately the point of the nail may actually penetrate the joint and it is then necessary to remove it, and if the fracture has not already united, to perform a Lorenz osteotomy

3 Ploughing of the nail through the head—The nail may not only penetrate more deeply but also plough upwards and forwards through the bone Comparison of antero posterior and lateral radiographs at two monthly intervals shows that the central position of the nail is not maintained, and that the head moves in relation to the neck into the position of *cova vara* (Figs 172 175) or *cova anteverta* (Figs 176 179) This must not be attributed to a fault in the nailing technique reinsertion of the nail or the introduction of a longer nail is a mistake Redisplacement is occurring because the bone is too fragile to withstand the pressure of a nail The correct treatment is removal of the nail and a Lorenz osteotomy

4 Slow union of the fracture—Slow union of the fracture despite the complete immobilisation of a well placed nail can be accepted as evidence of impairment of blood supply If union is not complete within about six months, the head is avascular If, for example, a nail has been removed after six or twelve months and refracture has immediately occurred, a second nailing operation or a nailing and grafting operation, is inadvisable (Figs 180 183) Similarly, if no cross pin fixation has been employed and extrusion of the nail has occurred with disengagement from the head after six months, followed immediately by redisplacement of the fragments, the nail should not be reinserted The head is avascular and necrotic, and an osteotomy should be performed

5 Late onset of arthritis—If the sign of relative density was not conclusive, and there has been no evidence of increasing penetration, ploughing of the nail or slow union, the final evidence of avascular necrosis is often delayed by many years A slowly developing degenerative arthritis may supervene five, ten or even fifteen years after fracture (Figs 184 185) Removal of the nail, or replacement of the nail by a bone graft, appears to have no influence on this late development (Even if performed at an earlier stage, a bone graft is of little or no importance as a conductor of blood vessels and a means of revascularising the head¹) If the symptoms are sufficiently pronounced to warrant it, arthrodesis of the joint is necessary

Treatment of old un-united fractures—The question as to whether a fracture several months old should be treated by nailing and grafting, or by a reconstruction operation, depends entirely on the question of avascular necrosis If there has never been evidence of impairment of blood supply, even a six or twelve months' old fracture may be successfully nailed If, on the other hand, there is proof of necrosis of the head, even a six weeks' old fracture should not be nailed or grafted The fragile bone will probably not hold the nail, and even if it does arthritis of the joint will supervene The type of

¹ D. B. Phemister Pathology of Ununited Fractures of the Neck of the Femur with Special Reference to the Head *Jour Bone and Joint Surg* 1939 **xxi** 681

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F. L. Compere and J. Lee: Restoration of Physiological and Anatomical Function in Fractures of the Neck of the Femur. *J. Bone and Joint Surg.* 194, xvii, 61.

2 **Increasing penetration of the nail and shortening of the neck**—Comparison of films taken at two monthly intervals may show progressive shortening of the neck and increasing penetration of the nail (Figs 169-171) The most reliable measurement is from the point of the nail to the articular surface Increasing penetration indicates abnormal fragility of bone and is evidence of necrosis It is most pronounced when early weight bearing has been permitted Ultimately the point of the nail may actually penetrate the joint and it is then necessary to remove it and if the fracture has not already united, to perform a Lorenz osteotomy

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5 **Late onset of arthritis**—If the sign of relative density was not conclusive, and there has been no evidence of increasing penetration, ploughing of the nail, or slow union, the final evidence of avascular necrosis is often delayed by many years A slowly developing degenerative arthritis may supervene five, ten or even fifteen years after fracture (Figs 184-185) Removal of the nail or replacement of the nail by a bone graft, appears to have no influence on this late development (Even if performed at an earlier stage, a bone graft is of little or no importance as a conductor of blood vessels and a means of revascularising the head¹) If the symptoms are sufficiently pronounced to warrant it arthrodesis of the joint is necessary

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¹ D. B. Flemister Pathology of Ununited Fractures of the Neck of the Femur with Special Reference to the Head *Jour Bone and Joint Surg* 1939 XXI 681



FIG 165 Two months



FIG 166 Four months



FIG 167 Eighteen months



FIG 168 Four years

Avascular necrosis with relative density of head, slow union of fracture and increasing penetration of nail

Nail fracture of the neck of the femur two months after operation shows relative density of the head (Fig 165). Collapse and disintegration of the avascular necrotic head was prevented by delaying weight bearing for eight months. Union was not complete until four years after operation (Fig 168). There was no increase in the penetration of the nail before weight bearing was permitted (Figs 165, 166) but some increasing penetration took place between the second and fourth years after operation (Figs 167, 168).

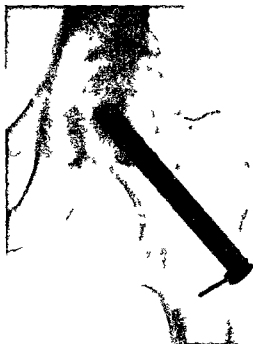


FIG 169 Three months



FIG 170 Nine months

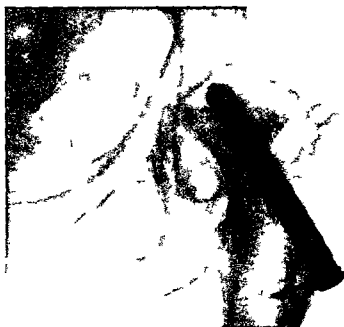


FIG 171 Two years

Avascular necrosis proved by increasing penetration of the nail

Radiographs three months after operation suggested the possibility of relative density of the femoral head but they were not conclusive (Fig 169). Weight bearing was permitted six months later there is clear evidence of increasing penetration of the nail, proving avascular necrosis of the head (Fig 170). Two years after operation the nail has penetrated the joint surface and the femoral head has integrated (Fig 171).



FIG 172 Three months



FIG 173 Four months



FIG 174 Five months



FIG 175 Three years

Avascular necrosis proved by ploughing of nail through the head (coxa vara)

Radiographs three and four months after operation show that the central position of the nail is not being maintained (Figs 172-173). The head has moved into the position of coxa vara. This proof of avascular necrosis was not recognised; it was thought that the nail was too short, a longer nail was introduced (Fig 174). Three years after operation the evidence of avascular necrosis is obvious in the sequel of degenerative arthritis (Fig 175). Removal of the nail has not arrested the development of the complication.



FIG 176 One week



FIG 177 Two months



FIG 178 Four months



FIG 179 Six months

Avascular necrosis proved by rapid ploughing of nail through the head (coxa anteverta)

Antero posterior and lateral radiographs one week after operation showed that the nail was centrally placed (Fig 176). Despite protection from weight bearing the nail ploughed through the front of the head (Fig 177). The head displaced backwards into the position of coxa anteverta (Figs 178-179). Even when the sign of relative density is inconclusive, re-displacement despite an accurately placed nail proves that the head is avascular, necrotic and fragile.



FIG 180 Three months



FIG 181 Eight months

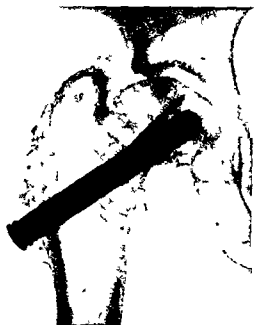


FIG 182 Six months after re-nailing



FIG 183. Eighteen months after re-nailing.

Avascular necrosis proved by slow union—early case illustrating many errors of treatment

Fracture of the neck of the femur treated by the original technique of open exposure of the fragments (first error). The nail is centrally placed (Fig 180). No evidence of relative density of the head was elicited. The nail was not driven home and the projecting head caused discomfort (second error). Eight months after operation it was thought that union was sufficiently sound for removal of the nail (third error). Within two days the fracture had redisplaced (Fig 181). The fact that slow union proved avascular necrosis was not recognised and the fracture was renailed (fourth error). One guide wire used for directing the nail broke off in the bone (Fig 182—fifth error). The certainty of avascular necrosis of the head was finally made clear eighteen months after re-nailing (Fig 183). The nail has ploughed through the necrotic head and the fragment of guide wire is penetrating the acetabulum. The first nail should not have been removed at the eighth month. Certainly when displacement recurred the fracture should not have been renailed. A Lorenz osteotomy was indicated. This was the treatment finally adopted despite the many disasters; the ultimate result was quite good.



FIG 184 One year



FIG 185 Eight years

Avascular necrosis proved by late development of degenerative arthritis

This patient resumed heavy work within twelve months of operation (Fig 184) and for several years the result appeared to be perfect. Eight years after nailing there is clear evidence of disintegration of the head and degenerative arthritis proving a slowly developing avascular necrosis (Fig 185)

reconstructive operation to be performed for old un united fractures is also to be determined by the problem of avascular necrosis. Many operations formerly described, such as the Brackett operation¹ in which the upper femoral shaft was implanted into the head, are now obsolete for this reason. Even the Whitman reconstruction operation,² in which the femoral head was removed and the stump of the neck implanted in the acetabulum, is likely to fail because stripping the neck deprives it of blood and induces avascular necrosis.³ The simple bifurcation osteotomy of Lorenz has superseded these procedures.

Traumatic Dislocation of the Hip Joint

The frequency with which traumatic dislocation of the hip joint is complicated by avascular necrosis has not hitherto been recognised. The sequel of degenerative arthritis is sometimes delayed for several years,⁴ and the dislocation and arthritis were not always associated as cause and effect. In fact, however, nearly every traumatic hip dislocation is complicated in this way to a lesser or greater degree.⁵ Rupture of the ligamentum teres causes localised necrosis of bone and cartilage in the region of the fovea in the majority of dislocations and in over 30 per cent of cases additional injury to capsular vessels causes complete disintegration of bone and severe degenerative arthritis.

Diagnosis of avascular necrosis in children—The complication may be recognised in early weeks by relative density of the upper femoral epiphysis which does not undergo disuse decalcification (Figs 186-190). Subsequent changes are those characteristically seen in Legg Perthes' disease. As revascularisation takes place and islands of decalcified bone appear, the epiphysis appears to be fragmented. If weight bearing is permitted the fragile bone is crushed, the femoral head is flattened and the epiphysis and neck are broadened (cova plana). Ultimately revascularisation is complete, the bone shows uniform calcification, but joint movement may remain limited by deformity of the head. Finally, ten or twenty years later, degenerative arthritis supervenes.

Treatment in children—The essential treatment is to protect the necrotic bone from compression until revascularisation and regeneration are complete. Early weight bearing is the worst treatment because it crushes the subchondral bone, distorts the joint surface, flattens the femoral head and interferes with regeneration of the articular cartilage. A plaster spica is of no particular value, and if the patient is allowed to walk in the plaster it is of no value at all. Even a walking calliper splint is ineffective. The patient must be recumbent and there must be traction on the limb. The traction should be continued until revascularisation is complete, and usually for eighteen months or two years. For the first few months, while the joint is irritable and there is muscle spasm with a tendency to flexion adduction deformity, the hip is also immobilised by means of an abduction frame. For

¹ Brackett. *Boston Med and Surg Jour* 1917, 35:1. *Jour Bone and Jo nt Surg* 1934 xx 83.

² R. Whitman. *Surg Gyn Obst* June 1911. *Jour Bone and Jo nt Surg* 1913 xv 215.

³ Plemister. *Aseptic Necrosis of Bone*. *Jour Bone and Jo nt Surg* 1930 xii 763.

⁴ E. N. Lott and B. E. Oblitz. *Aseptic Necrosis of the Head of the Femur following Traumatic Dislocation*. *Jour Bone and Jo nt Surg* 1939 xvi 301.

⁵ S. W. Banks. *Aseptic Necrosis of the Femoral Head following Traumatic Dislocation of the Hip*. *Jour Bone and Jo nt Surg* 1911 xxiii 37.

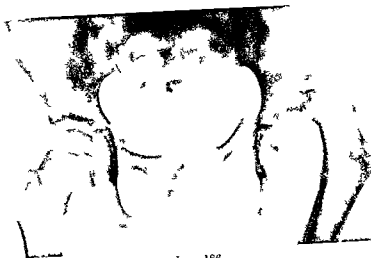


FIG 186



FIG 187 Three days



FIG 188 Four months



FIG 189 Ten months

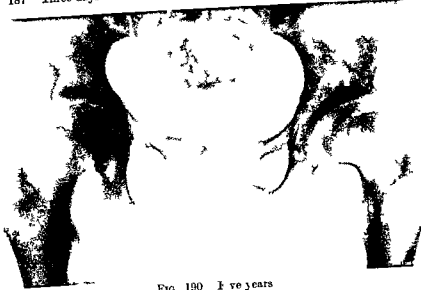


FIG 190 Five years

Dislocation of hip joint in child with avascular necrosis—(pseudo-coxa) Traumatic dislocation with fracture of pelvis (Fig 186) reduced by manipulation (Fig 187) Radiographs four months later show relative density of the area supplied by the vessels of the ligamentum teres (Fig 188) Typical changes of Legg-Perthes disease (pseudo-coxa) supervened (Fig 189) but coxa plana was maintained by prolonged recumbency and traction (Fig 190)

the next twelve or eighteen months traction is continued by weight extension, or by tying the limb to the raised foot of the bed and active hip movements are encouraged. When the apparently dense bone has passed through the fragmented stage to the stage of completely uniform calcification traction is discontinued. A further few months are spent in more energetic non weight bearing exercise for increasing periods each day. Finally after about two years unprotected weight bearing is permitted.

Diagnosis of avascular necrosis in adults—Every effort should be made to establish the diagnosis within two or three months of injury by the radiographic sign of relative density of the femoral head. If this evidence



Fig. 191

Location of hip joint—aicular necrosis and arthritis—minimal X-ray changes—narrow joint space
irregular density and irregular texture of head

disappearance of the joint space (Fig 192)

Treatment in adults—The treatment advocated in children namely, recumbency and traction for two years is seldom justified in adults. Not only is the adult less able to spend two years in bed but the regenerative powers of articular cartilage are less than in the child and it is unlikely that the sequel of early degenerative arthritis can be avoided. It is better to insist on recumbency for the first three or four months after injury and if severe arthritis with considerable pain and almost complete stiffness still develops to arthrodese the joint. The alternative of arthroplasty may be considered using the vitallium cup technique of Smith Petersen (Fig 193). But it is to be recognised that a hip joint soundly ankyllosed in the optimum position causes surprisingly little disability. There is no appreciable limp. It is easy to walk ten or fifteen miles without discomfort.

or fatigue, patients can jump, hop, climb and run, play tennis and golf, enjoy mountaineering and skiing, men go back to strenuous labour, women do their normal household duties, there is no pain in the back, there is little or no difficulty in sitting. The result is so completely reliable, that to compete with it an arthroplasty must be very perfect indeed. It is, however, essential that ankylosis shall be sound and that it shall take place in the optimum position of strictly neutral rotation, with neither abduction, adduction nor flexion deformity. A long three flanged nail must therefore be used transfixing the upper femur and pelvis¹ (Figs 194-195). Without the fixation of such a nail bony union cannot be relied upon, fibrous ankylosis occurs in nearly 50 per cent of cases and the result is unsatisfactory. Even with a nail union is seldom complete in less than twelve months or two years because the head of the femur is avascular; the nail is therefore essential in order to prevent recurrent deformity throughout this period². The case illustrated in Figs 196-198 proves the importance of nail fixation. A four months' old unreduced dislocation of the hip joint with sciatic palsy was reduced by open operation. Avascular necrosis was inevitable so that an arthrodesis was at once performed: the nail was driven into the pelvis at a second stage operation, two weeks after the first. The femur was fixed with just sufficient trace of abduction to correct true shortening. The head of the nail was not fixed to the femoral cortex with a cross pin, and some extrusion of the nail occurred. Eighteen months after operation the hip suddenly became painful, apparent shortening due to adduction deformity developed and radiographs showed that the nail had slid out of the pelvis. Although fixation had been maintained for no less than eighteen months the arthrodesis was still not firmly consolidated and the protection of a nail was still necessary. It was therefore reinserted in a new track and secured by a cross pin. Two years later consolidation of the arthrodesis was quite sound and the patient walked and ran excellently with no shortening, no deformity and a reliable painless hip.



FIG 192

Dislocation of hip with avascular necrosis and arthritis—marked X ray changes—gross irregular density and texture and disintegration of the head

¹ Watson-Jones. Arthrodesis of the Osteoarthritic Hip. *Jour Amer Med Ass* 1938 ex 274

² The operation of fixing the hip joint by means of a nail alone without preliminary denuding of articular surfaces, referred to in former editions of this book and described in *Jour Amer Med Assoc*, Jan 1938 ex 278, has been abandoned. The object of this procedure was to give the advantages of arthrodesis of the hip to old patients who were not fit for the full operation. Experience has shown that unless a hip joint is already almost completely stiff the fixation of a nail alone is not sustained. In the course of time painful movement develops, despite the nail. The excellent result which can be secured by arthrodesis of the hip in man is a two-stage operation—(a) denuding articular cartilage of acetabulum and femoral head (b) driving in a long three flanged nail under radiographic control.

the next twelve or eighteen months traction is continued by weight extension or by tying the limb to the raised foot of the bed and active hip movements are encouraged. When the apparently dense bone has passed through the fragmented stage to the stage of completely uniform calcification traction is discontinued. A further few months are spent in more energetic non weight bearing exercise for increasing periods each day. Finally after about two years unprotected weight bearing is permitted.

Diagnosis of avascular necrosis in adults—Every effort should be made to establish the diagnosis within two or three months of injury by the radiographic sign of relative density of the femoral head. If this evidence



Fig 191

Dislocation of hip with avascular necrosis and arthritis — normal ray angles — narrow joint space — irregular density and irregular texture of head

is inconclusive the complication should be suspected on clinical grounds if it is found that rotation movement remains limited and that flexion movement does not increase beyond 90°. In succeeding months the gradual development of flexion deformity confirms the diagnosis and as years pass by the range of movements becomes steadily less. The radiographic evidence may not be striking (Fig 191). Comparison with the normal hip shows reduction in the joint space due to erosion of articular cartilage and slight mottling of the head of the femur with irregular density and imperfect bone texture. In other cases the disintegration of bone is much more rapid the clinical diagnosis is obvious within twelve months or two years of injury and radiographs show gross distortion, crushing and irregular density of the femoral head with complete

disappearance of the joint space (Fig 192).

Treatment in adults The treatment advocated in children namely recumbency and traction for two years is seldom justified in adults. Not only is the adult less able to spend two years in bed but the regenerative powers of articular cartilage are less than in the child and it is unlikely that the sequel of early degenerative arthritis can be avoided. It is better to insist on recumbency for the first three or four months after injury and if severe arthritis with considerable pain and almost complete stiffness still develops to arthrodese the joint. The alternative of arthroplasty may be considered using the vitallium cup technique of Smith-Petersen¹ (Fig 193). But it is to be recognised that a hip joint soundly ankylosed in the optimum position causes surprisingly little disability: there is no appreciable limp; it is easy to walk ten or fifteen miles without discomfort

or fatigue, patients can jump, hop, climb and run, play tennis and golf, enjoy mountaineering and skiing men go back to strenuous labour women do their normal household duties, there is no pain in the back, there is little or no difficulty in sitting. The result is so completely reliable, that to compete with it an arthroplasty must be very perfect indeed. It is, however, essential that ankylosis shall be sound and that it shall take place in the optimum position of strictly neutral rotation with neither abduction adduction nor flexion deformity. A long three flanged nail must therefore be used transfixing the upper femur and pelvis¹ (Figs 194 195). Without the fixation of such a nail bony union cannot be relied upon. fibrous ankylosis occurs in nearly 50 per cent of cases and the result is unsatisfactory. Even with a nail union is seldom complete in less than twelve months or two years because the head of the femur is avascular the nail is therefore essential in order to prevent recurrent deformity throughout this period². The case illustrated in Figs 196 198 proves the importance of nail fixation. A four months' old unreduced dislocation of the hip joint with sciatic palsy was reduced by open operation. Avascular necrosis was inevitable so that an arthrodesis was at once performed, the nail was driven into the pelvis at a second stage operation, two weeks after the first. The femur was fixed with just sufficient trace of abduction to correct true shortening. The head of the nail was not fixed to the femoral cortex with a cross pin, and some extrusion of the nail occurred. Eighteen months after operation the hip suddenly became painful apparent shortening due to adduction deformity developed and radiographs showed that the nail had slid out of the pelvis. Although fixation had been maintained for no less than eighteen months the arthrodesis was still not firmly consolidated and the protection of a nail was still necessary. It was therefore reinserted in a new track and secured by a cross pin. Two years later consolidation of the arthrodesis was quite sound and the patient walked and ran excellently with no shortening, no deformity and a reliable painless hip.



FIG 192

Dislocation of hip with avascular necrosis and arthritis—marked X ray changes—grossly irregular density and texture and disintegration of the head

¹ Watson Jones. Arthrodesis of the Osteoarthritic Hip. *Joint Amer Med Soc* 1938 ex 24.
² The operation of fixing the hip joint by means of a nail alone with or without preliminary denuding of articular surfaces referred to in former editions of this book and described in *Joint Amer Med Soc* Jan 1938 ex 28 has been abandoned. The object of this procedure was to give the advantages of arthrodesis of the hip to all patients who were not fit for the full operation. Experience has shown that unless a hip joint is almost completely stiff the fixation of a nail alone is not sustained. In the course of time painful movement develops despite the nail. The excellent result which can be secured by arthrodesis of the hip demands a two-stage operation—(a) denuding articular cartilage of acetabulum and femoral head (b) insertion of a long three flanged nail under radiographic control.



FIG 193

Valtium cup arthroplasty for old unreduced dislocation of the hip
(Smith Petersen technique *Jour Bone and Joint Surg* 1931 xvi '69)



FIG 194



FIG 195

Artlrod s of hip for a sicular necrosis a l levererat ve artlrit s after dislocat o of the joint. A th e flanged nail must be used to ensure consolidat on n the opt mum post o. (Watson-Jones tech n que *Jour Amer Med Assoc* 1938 cx '78). The nail may be inserted at the t me of the artl o le s (Fig 194) or at a second stage opera on (Fig 195). A cross pin sho ld be sed to prevent extru on.



FIG 196 Three months



FIG 197 Nine months



FIG 198 Fifteen months

Unreduced dislocation of hip with avascular necrosis. Slow consolidation after arthrodesis of joint.

Four months' old unreduced dislocation with sciatic palsy. Avascular necrosis was inevitable and immediate arthrodesis was performed with second stage nail operation (Fig 196). The nail was not fixed by a cross pin and it gradually extruded (Fig 197). Even eighteen months later consolidation is unsound and when the nail finally left the pelvis pain and deformity recurred (Fig 198). The fixation of a nail is essential and extrusion must be prevented by a cross pin.

Legg Perthes' Disease of the Hip

Traumatic dislocation of the hip and transcervical fracture of the neck of the femur in children may produce changes which are clinically and radiographically indistinguishable from Perthes disease. They are indistinguishable because they are the same (but see p 78 footnote). This disease is simply an avascular necrosis of the upper femoral epiphysis. The frequency is explained by the vulnerability of the blood supply which may be cut off by the injuries we have discussed or by



FIG 199

Coxa plana (Perthes disease). Continued weight bearing has crushed the avascular epiphysis and caused flattening of the head and broadening of the epiphyseal line and neck.

the capsular trauma of manipulative and operative reduction of congenital hip dislocations. It may also be cut off by any strain or twist which causes thrombosis of capsular or ligamentum teres vessels or by the toxic and inflammatory changes which occur so commonly in the hips of children. Whether the source of deprivation of blood is obvious as in fracture or dislocation of the joint or concealed as in traumatic and toxic thrombosis the sequelae are the same.

Most typical cases of Perthes disease are already in the fragmented stage when they are first seen. Continued weight bearing has already crushed the epiphysis so that it is flat and broad. This accounts for the characteristic coxa plana and wide femoral neck (Fig 199). The appearance of density of the epiphysis and of fragmentation has been misunderstood.

We have believed that this was due to hypercalcification. Actually it is due to retention of the original calcium content while neighbouring bones have decalcified. If a case which ultimately develops the typical signs of Perthes disease is seen on the first day of limping the radiographic appearances are normal¹. If the hip is immobilised so that disuse decalcification arises and many radiographs are taken at weekly or fortnightly intervals one film will show failure of decalcification of the epiphysis (Fig 201). This stage is transient and may easily be overlooked.

The correct treatment of Perthes disease is the treatment of avascular necrosis of the femoral head in children. There must be complete freedom from weight bearing and continuous traction for twelve months to two years until revascularisation is complete. Immobilisation in a plaster spica is worthless and a walking caliper splint is of doubtful value. Since in many cases where there has been no actual dislocation or fracture the loss of blood supply has not been complete perfect recovery with a hip joint apparently indistinguishable from normal is often secured. Nevertheless as with any case of avascular necrosis of a joint surface the regenerated articular cartilage is imperfect and degenerative arthritis may arise ten or twenty years later.



FIG 200



FIG 201



FIG 202



FIG 203



FIG 204

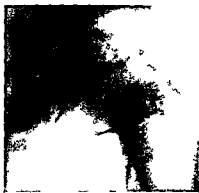


FIG 205

Perthes disease of hip on the first day of limping—no radiographic abnormality (Fig 200). Six weeks later failure of decalcification of epiphysis proving avascularity (Fig 201). Figs 202-205 at four monthly intervals show gradual revascularisation of epiphysis. Traction has been continued throughout and there is therefore no crushing or flattening.

AVASCULAR NECROSIS OF THE HEAD OF THE HUMERUS

The shoulder joint is dislocated much more commonly than the hip joint but the injury is the result of relatively trivial violence and the capsular damage is more localised. Shoulder dislocations are therefore seldom complicated by avascular necrosis of the bone. Furthermore fractures of the neck of the humerus seldom lie above all capsular attachments and the blood supply of the head of the humerus is usually preserved.

If there is a fracture dislocation of the joint the blood supply is more precarious. The head of the humerus is broken from the shaft and dislocated through a tear in the capsule. If every soft part attachment is torn the bone will undergo necrosis whether it is reduced or not. In one case a typical fracture dislocation of the shoulder was manipulated and the reduction appeared to be complete.¹ Post reduction radiographs showed that the head of the humerus was actually in the shoulder joint but it was upside down so that its fractured surface was in contact with the glenoid. At that time the significance of avascular necrosis was not recognised, an operation was performed and the head was tilted round into its proper position. The reduction was perfect but the loose head underwent complete necrosis and disintegration. The unnatural mobility which allowed the fragment to lie upside down was sufficient evidence to prove its complete detachment from soft parts. Avascular necrosis was therefore inevitable and the fragment might just as well have been left upside down. Ankylosis of the joint in the optimum position of moderate abduction was the best possible result. In such cases if bony fixation does not occur spontaneously an arthrodesis of the joint is advisable.

AVASCULAR NECROSIS OF THE ASTRAGALUS (TALUS)

Blood supply of the astragalus—The main blood supply of the astragalus enters the neck of the bone. There are large foramina on its dorsal surface



FIG 206

Blood supply of the astragalus. Vessels enter the neck on dorsal and ventral surfaces and also enter the body on its inner and posterior surfaces.

at the site of attachment of the anterior capsule of the ankle joint (Fig 206). Other large vessels enter its ventral surface from the interosseous ligament in the sinus tarsi. There are many smaller vessels entering

the body of the bone on its internal surface below the facet for the internal malleolus and posteriorly where the capsule of the ankle joint is attached to the trigonum

Fractures of the neck of the astragalus do not deprive either the head or the body of their blood supply (Fig 207). Even when this fracture is accompanied by dislocation of the subastragaloid joint so that all vessels entering from the sinus tarsi are destroyed there may still be an adequate blood supply from the dorsal and posterior vessels to both halves of the astragalus. But when the fracture is complicated by backward dislocation of the body of the astragalus all capsular attachments to the body of the bone are torn and it is entirely deprived of blood (Fig 208). Thus

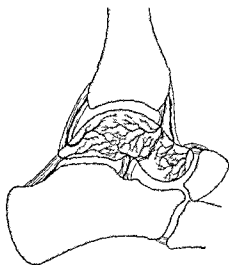


FIG 207

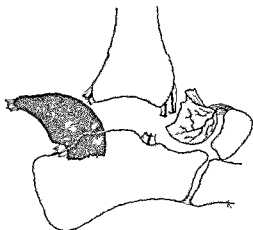


FIG 208

Fracture of the neck of the astragalus does not interfere with the blood supply to the body of the astragalus as long as the body is not dislocated backward (Fig 207). If the body of the astragalus is dislocated backward, the blood supply is cut off (Fig 208).

half of the astragalus undergoes avascular necrosis. If the displacement is reduced the fracture will unite and in the course of time the body of the bone is regenerated (Figs 209-213). Protection in plaster must be continued and the resumption of weight bearing deferred for many months in order to prevent crushing of the necrotic bone and degeneration of the articular cartilage. If degenerative arthritis does supervene it is serious because both ankle and subastragaloid joints are involved. Both joints must be arthrodesed. Arthrodesis of one of these two joints does not cause a very serious disability because function is taken over by the other and surprisingly free mobility remains. But if it is necessary to arthrodesise both joints the foot is rigid and inelastic. Nevertheless despite the rigidity the functional result of double arthrodesis is very much better than that of astraglectomy. After astraglectomy the foot is prone to develop inversion deformity and moreover it is weak and susceptible to strain.

Dislocation of the whole astragalus is a rare injury which may be followed by the same complication. A few soft tissue attachments may remain to preserve a sufficient blood supply. Every effort must therefore be made to



FIG 209
Dislocation of the body of the astragalus



FIG 210
Immediately after reduction

FIG 211
Six weeks after reduction

Figs 209-213 Fracture neck of astragalus with dislocation of the body, avascular necrosis and gradual revascularisation

Fracture dislocation of the astragalus with backward displacement and typical rotation of the body the subastragaloid articular surface faces outwards and the ankle joint surface inwards (Fig. 209). The dislocated fragment was replaced by manipulation (Fig. 210). Six weeks later there is evidence that the body is avascular because it has not participated in the decalcification seen in neighbouring bones (Fig. 211).



FIG 212

Six months after reduction

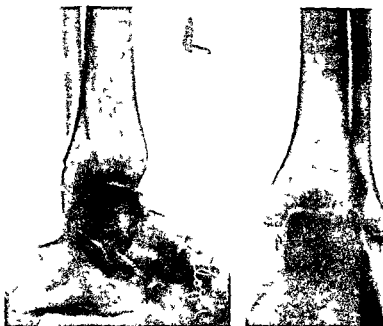


FIG 213

Twelve months after reduction

Immobilisation in plaster and protection from weight bearing was continued for many months. Serial radiographs showed gradual revascularisation proved by the development of areas of decalcification first in the region of the fracture and then spreading gradually backwards (Fig 212). The last area to revascularise was the subchondral bone and articular cartilage of the ankle joint. Both ankle and subtarsal joints show some narrowing of the joint space, indicating thin and imperfect articular cartilage (Fig 213). (The original fracture (shown) was reduced by Flight Lieutenant J. C. Adams).

reduce the dislocation by simple manipulation. If the manipulation fails it is probable that an operative reduction will damage any vessels which remain and again the problem is presented of rigidity of both ankle and subastragloid joints.

AVASCULAR NECROSIS OF THE CARPAL SCAPHOID BONE

Blood supply of the scaphoid—On the basis of the radiographic examination of scaphoid bones after injection of the vessels with opaque media it has been accepted that the arterial supply is from two main vessels, one entering the tubercle and one the waist.¹ If this is correct almost every fracture of the wrist and certainly every fracture of the proximal pole should deprive the proximal fragment of the scaphoid of its blood. We know from clinical observation that this is not so. Failure of decalcification of the proximal fragment is seen only in a proportion of proximal pole fractures and in a still smaller proportion of wrist fractures (Fig. 214).



FIG. 214

fracture waist of scaphoid the proximal fragment is completely avascular. It is no less dense than the upper shafts of the radius and ulna whereas the other carpal bones are decalcified.

Examination of a large series of scaphoid bones shows that the vascular foramina which are situated in the ligamentous ridge between the two main articular surfaces follow two types of distribution.* In two thirds of the bones the vessels are equally distributed throughout the length of the ligamentous ridge (Fig. 215). In the other third there are no vessels directly entering the proximal half. They pierce the cortex of the distal half and travel backwards in the middle of the bone. The foramina may be entirely confined to the tubercle or there may be a few small or one or two large foramina actually at the waist (Fig. 216). These facts explain the unequal rate of union of fractures of the tubercle, the waist and the proximal pole of the scaphoid and furthermore they explain the variable incidence of arthritis of the wrist after these injuries.

Fractures of the tubercle do not interfere with the blood supply of the fragments in either type of bone. The

fracture occurs in a very vascular region. It should therefore unite rapidly and with certainty. We find that this is so. Fractures of the tubercle always unite within a few weeks and non union is practically unknown.

Fractures of the proximal pole, on the other hand, must deprive the proximal fragment of its blood if the distribution of vessels is of the second type but not if it is of the first. This again coincides

¹ Iritzsky, Z. Anat. u. Chir., 1930, liv, 2600.

Obletz and Halbshtet, "Fractures of the Carpal 'Navicular'" Jour. Bone and Joint Surg., 1933, xx, 421.



Fig 21a



Fig 21b

Most commonly the blood vessels of the scaphoid are distributed throughout the length of the bone (Fig 21a). In one third of scaphoids the blood vessels all enter the distal half (Fig 21b).

Fractures
of the
tubercle



Fractures
of the
waist



Fractures
of the
proximal
pole



Fig 21c

With the first type of blood supply there is no danger of avascular necrosis from any fracture. It occurs only after dislocation. With the second type of blood supply necrosis of one fragment sometimes occurs in waist fractures and always occurs in proximal pole fractures.

with clinical experience. Two thirds of the fractures in this situation behave like fractures of the waist and unite promptly if immobilisation is adequate. In one third union is extremely slow. It may be necessary to continue immobilisation for twelve months or even longer and if immobilisation is not prolonged the fracture does not unite.

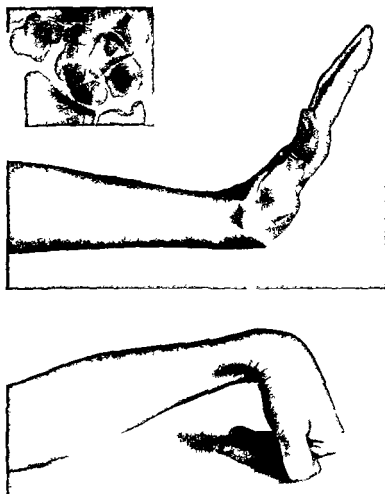


FIG 218

Fracture of scaphoid with avascular necrosis of the proximal half treated by early excision of the dead fragment. There is perfect movement and strength and complete freedom from pain.

Fractures of the waist usually unite with certainty after six or eight weeks of complete immobilisation. In two thirds of the cases the blood supply of the fragments is not even in danger. In the remaining third however the supply is precarious. The fracture may lie proximal to all vessels or it may damage and thrombose the one large artery which enters the wrist. Union will then be slow exactly as in proximal pole fractures.

Fracture dislocation of the scaphoid—If there is not only a fracture of the waist but also a dislocation of the proximal fragment there is a still greater likelihood of vascular impairment. Whatever the type of blood

supply it is in danger of being cut off by an actual dislocation. Slow union is therefore more common after fracture of the scaphoid with dislocation of the lunate and half scaphoid than it is after simple fracture.

Arthritis of the wrist after scaphoid fracture—We have seen that fractures can unite despite an avascular fragment if immobilisation is prolonged.



FIG. 219

Similar case to Fig. 218 treated by prolonged immobilisation. Despite firm union the wrist is very stiff and painful. There is arthritis involving the wrist and still more the mid carpal joint.

sometimes for twelve months to two years (Figs. 13-18). Slowness of union is not however the only penalty. If the fracture is not immobilised long enough for sound union the proximal fragment cannot revascularise and regenerate. Degeneration of the articular cartilage, arthritis of the joint and severe stiffness must supervene in succeeding years. Unfortunately even when prolonged immobilisation is rewarded by union of the fracture arthritis may still occur for the new fibro cartilage is imperfect. It follows that in any case where there is obviously complete loss of blood supply to one fragment this fragment should be excised.

Indication for excision of the scaphoid—If the proximal fragment is completely dislocated out of the wrist joint or if in any fracture after three or four weeks there is evidence of complete failure of decalcification of the proximal fragment it should be excised. When the operation is performed without delay an almost normal range of movement and a wrist joint little short of perfect can be secured (Fig 218). The decision must be made within a few weeks of injury. Delayed excision of the fragment is worthless. As the months pass by the articular cartilage of the radius and of the capitate opposite to the necrotic scaphoid fragment undergo secondary attrition and degeneration. Excision at this late stage fails to prevent the arthritis and indeed the trauma of operation may even precipitate the arthritis which it is designed to prevent.



FIG 220

Avascular necrosis of proximal half of scaphoid after fracture of the wrist. The wrong half of the bone has been removed.

that good function depends not simply on bony union but much more on freedom from avascular necrosis. Fig 219 shows the extremely limited range of movement in a case where necrosis and arthritis supervened despite firm union of the fracture. The result would have been much better if the fragment had been excised within a few weeks of injury, and the wrist not immobilised at all. *The fragment must however be excised at once and if both fragments are not excised it must be the dead half which is removed not the living half!* Fig 220 illustrates the haphazard indications of the older technique. The wrong half has been removed and the necrotic bone is still inflicting its damage on the joint.

AVASCULAR NECROSIS OF THE LUNATE BONE

Blood vessels reach the carpal lunate bone from both posterior and anterior ligaments and they enter through foramina on the dorsal and

Böller: The Treatment of Fractures. Vienna 1929.
 * Watson-Jones: Had quite in mobilisation and Non union of Fractures of Carpal Scaphoid Bone. Brit Med Jour 1914 May 6.

palmar interarticular surfaces. There are three types of dislocation of this bone, each interfering with the blood supply in varying degree (Fig. 221).

Retrolunar dislocation of capitate (os magnum)—In the first type the capitate is displaced backwards out of the cup of the lunate but the lunate itself remains normally attached to the radius by dorsal and palmar ligaments. The blood supply still reaches the bone almost normally and whether the dislocation is recent or old it should be reduced. Excellent results follow even delayed reduction.

Forward dislocation of the lunate—In the second type the dorsal ligaments of the lunate are torn and the bone is tilted forwards away from the radius and capitate. The intact anterior ligament carries a reduced blood supply. If the dislocated bone is promptly replaced by manipulation

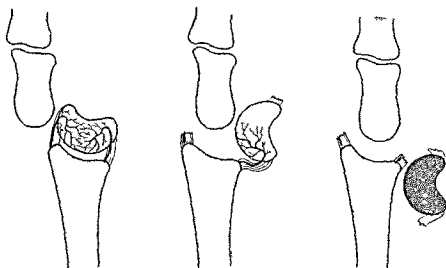


FIG. 221

There are three types of dislocation of the lunate. The blood supply is normal in the first, reduced in the second and cut off in the third.

it usually survives and a perfect result with complete freedom from arthritis may be expected. But if the dislocation is several months old, and especially if it can be replaced only by open operation, the reduced blood supply is still further impaired. An excellent anatomical position may be secured but the functional result will be very indifferent. In a series of twelve old dislocations of the lunate of this type which I reduced by operation some years ago there is only one perfect result. In all the others wrist movement is restricted and some are completely stiff. This was despite particular care and gentleness in operative reduction in view of the recognised danger of arthritis. Dissection of soft parts was minimised and a dorsal exposure was used in order to preserve the anterior ligament and the surviving blood vessels. Nevertheless avascular necrosis caused narrowing and arthritis of the mid carpal joint between the lunate and capitate and to a less extent of the radio carpal joint. These cases of old unreduced forward dislocation of the lunate are much better treated by excision.

Total dislocation of the lunate—In the third type both dorsal and palmar ligaments are completely ruptured and the bone which is denuded

of all soft part attachments is displaced into the lower forearm. Avascular necrosis is inevitable. Traction and manipulation may succeed in reducing the dislocation. Heroic methods of metacarpal skeletal traction with pins and wires have been described. But successful reduction does the patient a disservice. The X ray appearances are possibly more attractive than after excision but the joint will become stiff, painful and susceptible to strain. Function is vastly superior after early excision. Even if the case is seen on the first day after injury, the widely displaced lunate which is obviously deprived of all its vessels should be excised.

AVASCULAR NECROSIS OF THE EXTERNAL CONDYLE OF THE HUMERUS

Children sometimes sustain a fracture of the external condyle of the humerus and the fragment is completely tilted and rotated away from the main fragment. The common extensor origin of muscles and the outer capsule of the elbow joint remain attached to the loose fragment and are responsible for its blood supply (Fig. 222). If these soft tissues are dissected from the bone in the course of an operative reduction, avascular necrosis will supervene. The fragment carries two thirds of the articular surface of the humerus and very serious restriction of elbow movement remains permanently. Every attempt must be made to reduce these fractures by manipulation. If manipulative reduction fails, operation should be undertaken with caution. Difficult as it may be, the fragment must not be cleared of soft part attachments. Every muscular or capsular fibre arising from it is to be preserved and the reduction must be carried out with the utmost gentleness. If reduction is successful and the blood supply is not impaired, the result will be perfect. If the blood supply is destroyed and necrosis develops, there will be little or no movement below the right angle. This disastrous result cannot be improved upon by excision of the fragment for lateral instability, cubitus valgus deformity and delayed ulnar palsy will supervene. It is therefore imperative that no effort should be spared in preserving the blood supply of the fragment.

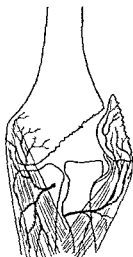


FIG. 2

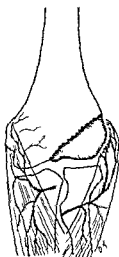


FIG. 3

The displaced external condyle of the humerus depends for its blood on the capsule and muscles attached to it. If these are dissected off during operative reduction, necrosis of the condyle and stiffness of the elbow joint supervene.

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AVASCULAR NECROSIS AND OSTEOCHONDRITIS DISSECANS

Any intra articular fracture which completely separates a fragment of bone and articular cartilage causes necrosis of the detached fragment. Small fragments of the capitellum may be detached by the impact of the

head of the radius which itself sustains a marginal fracture. The capitellar injury is often overlooked but there can be little doubt that avascular necrosis of the articular cartilage explains the limited elbow movement so common after fractures of the head of the radius. These injuries must be regarded with suspicion. Good results are possible only if detached and loose fragments are removed at once. Delayed excision of the radial head will not relieve the stiffness of the elbow for the arthritis following necrosis of the fragments is then already developed. Similarly fragments of the articular surfaces of the knee joint may be broken off and undergo necrosis. The external tuberosity of the tibia is sometimes comminuted by the impact of the outer femoral condyle forced into the tuberosity by extreme valgus deformity. There is a large outer marginal fragment of the tuberosity which preserves its soft tissue attachments and its blood supply and several smaller completely loose fragments lying between it and the main bone. The cartilage of these fragments will necrose and they should not as a rule be elevated to the joint surface. It is better to replace the main marginal fragment which is alive and to leave a central crater of depressed dead fragments. If the fragments are displaced between the joint surfaces they should be removed not replaced great care being taken to preserve all soft tissue attachments to the marginal fragment of the tuberosity.

In other cases fragments of the femoral condyles become separated and necrotic without a clear history of injury. This is the condition known as osteochondritis dissecans. The pathology is undoubtedly that of avascular necrosis and it seems certain that there is usually a traumatic factor. The lesion always occurs in those areas of the knee elbow and ankle joints which are susceptible to trauma and where true fractures of the joint surface are often seen. But thrombosis of the end arteries which supply these fragments of bone and articular cartilage may perhaps occur from other causes. One patient within the short period of two years developed osteochondritis dissecans in the femoral condyle of one knee the patella of the opposite knee the external condyle of one elbow and the head of the radius of the other elbow. It is difficult to believe that there is not a general constitutional factor in such a case.

VASCULAR INJURIES COMPLICATING WOUNDS AND FRACTURES

This courageous observation was made by Hunter 170 years ago¹ after studying the contraction of the muscular coat of arteries in response to injury. When he divided the blood vessels in the thigh of a boar bleeding ceased before the animal weakened. After exposure of the posterior tibial artery of a dog the vessel "was observed to be so much contracted in a short time as almost to prevent the blood from passing through it and when divided the blood only oozed out from the orifice." Hunter's work was the first recognition of segmental spasm of arteries the vasoconstriction which protects against rapid and complete exsanguination after severe wounds. His observations have been confirmed by later clinical experience. Makins² recorded cases in the last war of large arteries severed by bullets without external hæmorrhage or hæmatoma formation. In this war an airman's foot was severed at the ankle joint by the propeller blade of an aircraft he was admitted to hospital in excellent condition having suffered little loss of blood. A commando soldier under training sustained a violent hyperextension strain of the knee and complained of tearing pain in the popliteal space operative exploration showed complete rupture of the popliteal artery with little more than a teaspoonful of blood in surrounding tissues. Holdsworth³ has reported two cases of avulsion of the whole upper limbs including the clavicle and scapula in which there was no arterial bleeding. These cases could be multiplied. They illustrate the fundamental principle that arteries respond to injury by traumatic arterial spasm.

Traumatic arterial spasm—Spasm of the muscular coat of an artery occurs not only after severance and rupture but also after contusion or even concussion of the vessel. The disruptive force of a bullet or shrapnel traversing the tissues near an artery but not actually producing a demonstrable lesion of the vessel may cause segmentary spasm (*spasme arterielle* or Kroh's arterial spasm as it was called by English, French,^{4,5} and German⁶ observers in the last war. Kroh described a revolver bullet wound in the region of the femoral artery which arrested the circulation of the limb. At operation the artery was found apparently undamaged but contracted to

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the size of a knitting needle to the surgeon's surprise while the artery was exposed it dilated to normal size. Cohen¹ records a recent case of a penetrating wound of the elbow due to a bomb casing splinter with absent radial and ulnar pulses the brachial artery was so contracted as to be smaller than adjacent venæ comites, although actual injury was limited to fine stippling of its wall simple exposure of the artery and separation from its bed of areolar tissue was enough to relieve the spasm.

Traumatic arterial spasm may also occur when there is no external wound. The artery may be contused by one fragment of a fractured bone and this is now recognised with increasing frequency as a cause of ischæmic contraction and gangrene. The brachial artery is in danger in supracondylar fractures of the humerus the radial and ulnar arteries in fractures of the forearm (Figs 224-225) the axillary artery in fractures of the neck of the humerus and the popliteal artery in high oblique fractures of the tibia (p. 128). A violent blow which does not fracture the bone may cause traumatic arterial spasm and gangrene as for example in bumper injuries of the knee where the tibial artery is contused against the neck of the fibula.² Counterpressure on the popliteal vessels by the cross bar of a Böhler's traction frame may be responsible.³ Similarly the sharp pressure of a ridge of plaster may traumatise an artery as reported by Trueta⁴ in a case where plaster was applied to an elbow in extension a few moments later an assistant walked in and believing that a mistake had been made proceeded to flex the joint before the plaster had set" thus buckling the cast in the fold of the elbow. The circulation was completely arrested and at operation the brachial artery was found reduced to one third its normal size. Many cases are now recorded of the tight application of tourniquets causing arterial spasm which persists long after the tourniquet has been released. In one case the femoral artery remained pulseless eighteen hours after the removal of a tourniquet which had been left on the limb for six hours.⁵ Exposure of the artery showed no hæmatoma and no local internal laceration but only spasm which was relieved by division of the fascial roof of Hunter's canal and mobilisation of the artery. Esmarch introduced his flat rubber tourniquet sixty five years ago because gangrene had followed the use of inelastic tourniquets but even Esmarch's tourniquet is not immune from the danger. Vasospasm of the brachial artery causing gangrene of the forearm and hand has occurred from the pressure of an Esmarch rubber tourniquet at the lower axillary margin for only forty five minutes (Fig. 246).

Spasm of the artery is not limited to that part of the vessel which sustains injury it also involves the main branches the vessels of the collateral circulation and sometimes the trunk of the artery proximal to the level of injury. Clearly therefore it is more than a local myogenic reaction of injured tissue in the wall of an artery.^{6,7} It is almost certainly a reflex vasoconstriction the afferent impulses arising from the damaged region of the artery passing to the paravertebral plexus and stimulating the whole sympathetic nerve supply of the affected limb.⁸ Experimental evidence

¹ Cohen *Lancet* 1941 807 and *Guy's Hosp. Rep.* 1940 xc 901.
² G. Jones *Amer. J. Surg.* 1939 xl 1 290.
³ M. Cohen *Guy's Hosp. Rep.* 1940 xc 11.
⁴ J. Trueta *J. Bone & Joint Surg.* 1934 16 198.

⁵ D. L. Crisfield *Brit. Jour. Surg.* 1940 xx iii 6.
⁶ Kuttner and Baruch *J. ex. Med.* 1940 190.
⁷ J. A. McEwen *Proc. Roy. Soc.* 1940 13x 109.
⁸ R. Leticic *Ann. of Surg.* 1940 1xxviii 440.

suggests that it may even overflow to the opposite limb and cause vasospasm in the main artery on the uninjured side.¹ The reflex character of the spasm is suggested not only by its widespread distribution, but also by the relief



FIG 224

Normal arteriogram



FIG 225

Arteriogram in ischaemic contracture

Vascular occlusion due to fracture

Arteriograms after injecting perabrodil in a case of shrapnel wounds without vascular injury (Fig 224) and of ischaemic contracture after fractures of both forearm bones (Fig 225). The radial artery has been contused by the fracture of the radius and the interosseous artery by the fracture of the ulna—the ulnar artery is intact. (By courtesy of Mr Bremner-Hughes see p. 133.)

which usually follows removal of the source of afferent impulses by resection of the injured segment of artery,² and which may follow cutting the sympathetic reflex arc by paravertebral novocaine injection³ spinal or brachial

¹ Barnes and Trueta *Brit Jour Surg* 1947 xxx 74
² R. Leriche and Wierquin *Lancet* 1940 ii 290

³ J. C. White *The Autonomic Nervous System*
 New York, 1935

plexus anaesthesia, or operative sympathectomy.¹ A dramatic case illustrating these features of traumatic arterial spasm—the minor degree of injury, the widespread distribution of spasm and the favourable response to excision was reported by Griffiths.² A boy of seven sustained a supracondylar fracture of the humerus. Before reduction of the fracture the hand and forearm were normal but immediately after the manipulation they changed dramatically becoming suddenly blanched cold, pulseless and anaesthetic. I explored the arm and found a small hæmatoma no larger than a millet seed in the wall of the brachial artery at the site of fracture. Despite the smallness of this hæmatoma the whole artery and its radial and ulnar branches were collapsed and pulseless from the insertion of the coraco brachialis downwards. The collateral vessels were equally collapsed—the anterior ulnar recurrent was the size of a retinal artery. Mobilising and stripping the brachial artery produced no change but after resection of the damaged portion the arm made a complete recovery. Circulation was fully restored, and there was neither gangrene nor contracture.”

The underlying purpose of reflex vasospasm is no doubt to protect an animal from bleeding to death after severe arterial injury, but for this protection the animal must pay the price of ischaemic contracture or even gangrene. The spasm usually persists for at least twenty four hours, and sometimes for three or four days. The distal pulse may then return, but it is too late, the damage has been done, the muscles are necrosed and despite the return of a strong pulse the limb may still become gangrenous. Treatment is therefore urgent. If grave complications are to be avoided the circulation must be restored within six or eight hours. Non operative measures may be tried including the intravenous injection of antispasmodics,³ paravertebral novocaine injection of the sympathetic ganglia⁴ or interruption of the vasoconstrictor fibres by brachial plexus block, or spinal anaesthesia. The most certain and reliable treatment is early exposure of the artery, separation from its bed and the removal of surrounding areolar tissue. Hot saline lavage of the wound or massage of the artery may assist. If spasm still persists, arteriotomy is advisable, particularly when the wall of the artery shows actual bruising.

Traumatic venous spasm—It is to be noted in passing, that vasospasm is not limited to the arteries but may also involve the veins of a limb. In thrombophlebitis, constriction of the veins occurs in the collateral venous network and gives rise to oedema which could not be explained by simple occlusion of one vein. Moreover thrombophlebitis also causes arterial spasm, which disappears on novocaine infiltration of the lumbar sympathetic.

Arterial contusion, thrombosis and embolism—Contusion of an artery may obstruct the lumen of the vessel by causing thrombosis at the site of a

¹ M. Cane and A. Oelner, *Lancet*, 1940, vol. 93.

² D. L. Griffiths, *Brit. Jour. Surg.*, 1940, xviii, 239.

³ I. A. van der Horst, *Hydrochloride* (1 gr.) is injected intravenously. If vasodilatation does not occur, and there are no untoward effects a second dose of 1 gr. may be given in thirty minutes. *Asy. Soc. An. Affen. Collecte. Papers*, Mayo Clinic, 1936, xviii, 647.

⁴ Injection of the sympathetic ganglia with 2 per cent. novocaine at the 2nd and 3rd thoracic intervertebral spaces for the upper limb and the 4th and 5th lumbar spaces for the lower limb is strongly advocated by J. C. White (*The Autonomic Nervous System*, New York, 1933). Needles 10 cm. in length are inserted through the skin 3 cm. lateral to the upper margin of each spinous process, perpendicular inward to a depth of 4 to 5 cm. until they touch the transverse process. An then directed slightly upwards and inwards and thrust 4 cm. through the pons until the side of the vertebral body is felt. In order to exclude the dangers of injecting into an artery or the subarachnoid space the needle should be inserted independent of the syringe only fitted to them after they are in position and aspirated in attempt to detect novocaine. In inject. 1. Two cubic centimetres of 2 per cent. novocaine is then injected at each level. Rapid warming and drying of the foot proves that the needles have been accurately placed.

crack in the tunica intima. The circulation of the limb is often maintained by the aid of collateral vessels, but sometimes it is further obstructed by distal extension of the thrombus or by the release of an embolus which impacts at a lower level, at the opening of a large branch, or at a site of bifurcation. Moreover, the irritant effect of a thrombus or embolus within an artery is itself sufficient to induce secondary arterial spasm and cause further embarrassment to the circulation. Secondary vasospasm may indeed be so widespread as to confuse the clinical signs and create difficulty in establishing the level of obstruction. For example, in one case, a small embolus lodging in the posterior tibial artery gave rise to reflex vasospasm involving both legs, thus resembling the clinical picture of obstruction at the bifurcation of the aorta.¹ The cumulative effect of thrombosis, embolism and reflex vasospasm leading ultimately to gangrene is illustrated in Figs 226-228 a case of an ununited fracture of the clavicle with repeated contusion of the subclavian artery over a period of four years. Whenever the shoulder was abducted to the right angle the weight of the limb forced the outer fragment of the clavicle inwards so that it impaled the artery against the first rib. After four years of vasomotor instability with alternating pallor, redness and cyanosis of the hand, gangrene of the index and middle fingers developed. A cervical preganglionic sympathectomy was performed, and ten days later the ununited fracture was bone grafted. Pain was relieved, the hand became warmer than it had been for years and progress of the gangrene was arrested. The patient died later of a streptococcal throat infection. Post mortem examination of the subclavian artery showed roughening of the tunica intima at the site of bony impact. Thrombi formed at this level had become detached and impacted as emboli at the bifurcation into axillary and profunda branches. Both vessels had been occluded for a long time and were replaced by solid cords of mature fibrous tissue. The final circulatory failure was due to vasospasm of the collateral vessels, caused by continued contusion of the artery, and relieved by cervical sympathectomy.

The dangers of spreading thrombosis, embolism and reflex vasospasm make it clear that the correct treatment of thrombosis of a contused artery is to expose the vessel, ligate it above and below the thrombus and excise the thrombosed segment. This was recognised from the experience of 1914-18. One of the rules of the official "History of the War" reads: "When a large vessel is exposed in an open wound and has obviously suffered contusion and is thrombosed the vessel should be ligated above and below the thrombosed segment and the latter excised." Experience since the last war shows that this rule applies not only in the case of open wounds but also in closed injuries of large vessels where contusion by the fragments of a fractured bone may cause thrombosis.

Rupture and perforation of arteries—Rupture or perforation of an artery may be a primary injury due to the penetration of glass, shell or bomb fragments or other foreign bodies driven into the tissues with high velocity (Figs 229-232). Even when there is no external wound, an artery is sometimes perforated by the sharp spicules of a fractured bone. Late rupture with secondary hæmorrhage is usually due to erosion of the wall of a vessel which is exposed in an infected wound, but this also may occur in closed

¹ L. N. Atlas. *Surg. Gyn. Obst.* Feb. 1942, lxvii, 231.



110 296

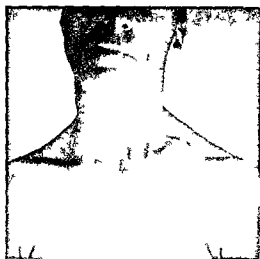


FIG 7



FIG 95

Arterial contusion causing embolism and vasospasm

Ununited fracture of clavicle with repetitive contusion of the subclavian artery by the outer fragment causing thrombosis, embolism and vasospasm. At the site of contusion (top of the specimen) there is roughening of the tunica intima. Embolus detached from this site has blocked both main brachial arteries. Continued contusion of the periarterial sympathetic nerves caused vasoconstriction of collateral vessels and finally gangrene of two fingers.

injuries as the result of simple contusion and necrosis of the damaged wall of the vessel. If there is an open wound external hæmorrhage causes shock or death, if there is no wound internal hæmorrhage into surrounding tissues leads to a pulsating hæmatoma and traumatic aneurism. In the treatment of these cases reliance cannot be placed on traumatic internal spasm for the control of hæmorrhage. It is true that serious bleeding from a completely severed artery ceases within a few minutes even when no ligature has been applied but protective vasospasm may not persist for more than twenty-four hours and the control of hæmorrhage then depends upon a recent and not very firm thrombus. Moreover a tangential wound or perforation which does not completely sever the vessel cannot be sealed by spasm of the muscular coat. In experimental cases although bleeding from a severed artery ceases within a few minutes bleeding from a tangential wound continues until death of the animal. A large bleeding vessel in an open wound must therefore be ligatured at the first possible moment. Even if the vessel is not actually bleeding at the time of operation it must be ligatured. If the ends of the artery are retracted and out of sight it is still essential to find them and apply ligatures.

Secondary hæmorrhage—Secondary hæmorrhage in infected wounds must be controlled by exploring the wound and finding the bleeding vessel and not by ligation of the main artery at a more proximal level. Proximal ligation of the main artery often fails to control the bleeding it adds the risk of gangrene of the limb and by reducing the oxygenation of tissues it lowers the defence of the wound to infection and particularly anaerobic infection. The surgeon may be faced with great difficulties. Hæmorrhage is sometimes so brisk that he cannot see the wound fills with blood faster than a dab can be withdrawn he is tempted to plunge hæmostatic forceps into the bloody pool and clamp blindly whatever comes within reach. One young resident surgical officer far exceeding his duty in attempting the operative reduction of a late unreduced dislocation of the shoulder divided a strong adhesion. Before the bleeding from a severed axillary artery was controlled he had irreparably crushed all the trunks of the brachial plexus. The solution to the problem of finding the bleeding point in a rapidly filling pool of blood is the use of many gauze packs firmly pressed into the wound and left undisturbed for several minutes. The incident recorded in the footnote below¹ illustrates the relative ease with which forceps can then be applied.

Dangers of ligation in continuity—When division of an artery is incomplete the vessel must not be ligated in continuity. Ligatures must be applied above and below the level of perforation the injured segment then being excised. This is necessary not only to avoid reflex vasospasm but also to prevent subsequent erosion of the vessel wall. When a large artery is ligated in continuity the constant battering of pulse beat is applied to a fixed point and late erosion with secondary hæmorrhage is always a danger on the

¹ A surgeon in France, visited by members of a travelling surgical club, was performing a nephrectomy. One of his assistants revealed the surgeon's reputation for dealing successfully with the problem of the ligature slipping off the renal pedicle. "What was the secret?" "I will show you," was the reply, as he cut off the kidney with a pair of scissors without first applying either forceps or ligature to the renal pedicle artery or vein. "There is the problem," he said stuffing large gauze packs tightly into the cavity. "He is the operating table," commented on the weather, discussed the political situation and drank a cup of tea. Five minutes passed before he returned to the task. On gently removing the packs the field remained dry, a te long enough for him to apply hæmostatic forceps with great deliberation to both artery and vein. His secret lay in the exercise of restraint and in allowing sufficient lapse of time for the hæmorrhage to be controlled by a sustained pressure and reflex vasospasm.



FIG 229

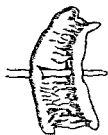


FIG 230

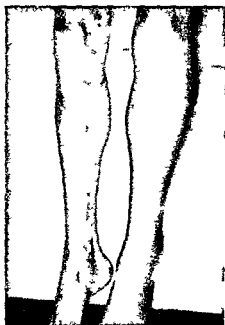


FIG 231



FIG 232

Perforation of popliteal artery arterial hæmatoma and ischaemic contracture

The popliteal artery was perforated by a shell fragment the size of a pinhead (seen in the middle of the thigh Fig. 229). The calf was diffusely swollen by a pulsating arterial hæmatoma (Fig. 231). The injured segment of artery was excised. Fig. 230 shows the wound is of entry and exit. Gangrene as well as ischaemic contracture supervened (Fig. 232).

(By courtesy of M. S. C. See page 134.)

other hand, section of the vessel allows elastic retraction and dissemination of the force of the pulse beat

Simultaneous ligation of vein—The danger of gangrene after ligation of the main arteries of the limbs is greatest in the case of the common femoral and the popliteal arteries. The risk is less when the accompanying vein is simultaneously ligatured. This has been confirmed by experimental evidence¹ by clinical studies^{2,3} and by the report in 1917 of the Inter Allied Conference of Surgeons in Paris which recommended "that the ligation of a large artery for injury should be accompanied also by occlusion of the satellite vein even although the latter be uninjured".⁴ The exception to this rule applies in the case of the 'Henle Coenen phenomenon'⁵, if after ligation and division of an artery the distal stump is seen to pulsate, it is clear that the collateral circulation is already adequate, and simultaneous ligation of the accompanying vein is then unnecessary.

Suture of artery—Successful suture of ruptured blood vessels has become possible since the introduction of heparin by which thrombosis can be controlled. Heparin in saline is injected into the repaired vessel proximal to the line of suture and continued after operation by intravenous infusion for four or five days.⁶ End to end anastomosis is performed with fine silk on eyeless needles using a continuous vertical mattress stitch which everts and approximates the edges and exposes a minimum of thread to the lumen of the vessel. Drying of the vessel during suture is prevented by dropping sterile olive oil into the open ends. It must be recognised, however, that the suture of ruptured vessels is still on trial. Results in the last war were disappointing not only because no method was then available for the control of post operative thrombosis but because even when this danger was avoided the scar in the vessel wall subsequently gave way in the majority of cases and aneurisms developed necessitating excision of the sac and ligation of the vessels.⁷

Pulsating hæmatoma and traumatic aneurism—If a perforated artery does not communicate with an open wound, bleeding into surrounding tissues produces an arterial hæmatoma. After several weeks or months the hæmatoma resolves, pulsation develops and a traumatic aneurism is formed. Rupture of an artery is occasionally due to simple contusion. A surgeon who was playing cricket stopped a fast ball in the palm of his hand. After two hours there was tingling, followed by intense pain in the distribution of the median nerve. The lightest touch on the thumb, index or middle fingers or even a breath of air was sufficient to precipitate agonising waves of pain. The hollow of the palm was filled and the front of the wrist tightly swollen. Aspiration of 16 c.c. of blood gave temporary relief, but within twenty four hours paralysis of the median nerve was complete. Operation disclosed rupture of the superficial palmar arch with an arterial hæmatoma filling the space beneath the annular ligament and infiltrating

¹ F I Reichert, *P H Johns Hopkins Hosp* 1931 xlix 56

² I Seibert, *Met Klin Berl* 1916 xli 1338. A series showing gangrene in 20 per cent. of lower limb ligatures of artery alone and only in 9 per cent. of ligation of artery and vein. And in the upper limb gangrene in 7.5 per cent. of ligatures of artery alone and in no cases of ligation of artery and vein.

³ L H Little, *I et z Klin Chir* 1921 cxxix 60. A review of 292 ligatures of large arteries alone with gangrene in 154 (52.5 per cent.) and of 194 ligatures of both artery and vein with gangrene in 17 (8.5 per cent.).

⁴ J Leimbarton and J J McLaughlin, *Ann of Srg* 1917 xvi 1103

⁵ H Best, *Brit Med Jour* Nov 1934 977. From 20 to 100 mg. of heparin in 20 c.c. of saline is injected into the vessel and an intravenous drip containing 10 mg. heparin per 100 c.c. saline is continued after operation at the rate of 70 drops a minute.

⁶ G Murray, *Lancet* July 13 1935 433

⁷ I H Mitchell in, *N Thoms Hosp Gaz* London 1940 xxxviii 99. *Brit Med Jour* July 1939 124

the lumbrical muscle bellies. Ligation of the vessels and division of the annular ligament gave immediate relief from pain but several months elapsed before the median paralysis recovered.

Perforation of an artery may also be due to the penetration of a sharp spicule of bone. Figs 233-235 show a comminuted Colles' fracture of the radius complicated by perforation of the radial artery and a traumatic



FIG. 233

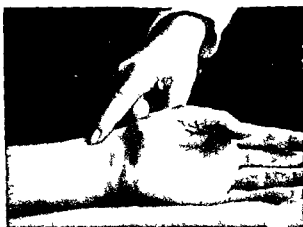


FIG. 234



FIG. 235

Traumatic aneurism

Aneurism of the radial artery due to puncture of the vessel by a bone spicule from a comminuted Colles' fracture

aneurism which was first recognised several weeks after the plaster had been removed. The femoral artery is sometimes perforated in the upper thigh by one fragment of a fractured shaft of the femur. The circumference of the thigh is increased several inches by a deeply fixed swelling which is extremely hard which may slowly increase in size does not at first pulsate and may almost present the clinical picture of a sarcoma of bone. After several months as the laceration undergoes resolution the development of expansile pulsation and a systolic bruit make the diagnosis clear. Regular measurements of the circumference of the limb should be recorded. If the

hæmatoma is not increasing, and there is no threat of ischæmia or gangrene, operation should be deferred long enough to assist the establishment of a collateral circulation and to reduce the difficulties and danger of operation. A delay of two or three months is advisable. Whenever possible, the usual Hunterian ligation of the artery proximal to the aneurism should be supplemented by distal ligation and excision of the intervening segment. Before the operation is performed, special care must be taken to exclude the possibility of venous as well as arterial communication with the hæmatoma. If a venous communication is overlooked and an arteriovenous fistula is treated by proximal ligation of the artery alone the result is utterly disastrous.

Arteriovenous fistula.—Simultaneous perforation of artery and vein gives rise to an arteriovenous fistula. The communication may be direct (aneurismal varix) or through an intervening false aneurism (varicose aneurism). The diagnosis is established by the development of a thrill and bruit which is continuous throughout the cardiac cycle. Continued escape of arterial blood into the veins and right side of the heart calls for a greatly increased cardiac output in the attempt to maintain normal arterial pressure. If the fistula is large more than half the circulating blood may leak back into the capacious venous bed. Dyspnœa and tachycardia arise on the slightest exertion, attacks of momentary faintness develop, there is an increasing "pounding" of the heart, slowly developing decompensation due to cardiac dilatation and ultimately invalidism and death. Occasionally there are local manifestations in the area of venous back pressure, the veins may become enormously dilated, cedema is almost elephantiasis in type and there is varicose eczema and ulceration.

The differential diagnosis between a simple traumatic aneurism and an arteriovenous aneurism is of the greatest importance, because whereas proximal ligation of the artery alone may suffice for a simple aneurism this operation fails to cure an arteriovenous aneurism or to relieve the cardiac distress and it usually causes gangrene of the limb. Blood finds its way through the collateral circulation of the ligated artery back to the fistula and vein and only to the limb beyond the fistula with even greater difficulty than before. The distinctive features of a fistula as compared with an aneurism are (1) the thrill and bruit are continuous but intensified in systole in the fistula and occur only in systole in the aneurism, (2) digital closure of the artery proximal to the lesion slows the pulse rate and increases the blood pressure in the presence of fistula, but never in the presence of simple arterial aneurism.

Early operation shortly after injury is needed only when hæmorrhage from the wound continues or when an increasing hæmatoma threatens to obstruct the circulation of the limb. The wound is then laid open, blood clot is removed, debridement is performed and both artery and vein are ligated and divided. Free drainage is established by leaving the wound widely open. As a rule these aneurisms develop gradually, and several weeks elapse before they are clinically obvious. In these circumstances provided that cardiac decompensation is not threatening operation should be deferred until at least three months after injury. This delay assists the development of a collateral circulation and makes the operation easier and more safe. Complete rest in bed for two weeks before operation is advisable. digital

closure of the fistula for thirty minutes three to six times a day may also assist in controlling cardiac decompensation. The operation of choice is ligation of the artery and vein proximal and distal to the fistula with excision of the fistula.¹

THE RESULTS OF ARTERIAL INJURY

The types of arterial injury which have been considered include (i) contusion of an artery causing traumatic arterial spasm (ii) contusion of an artery causing thrombosis at the level of injury or embolism at a more distal level (iii) perforation or rupture of an artery causing external hemorrhage (iv) perforation or rupture of an artery causing internal hemorrhage arterial hæmatoma and traumatic aneurism or arteriovenous fistula. Any of these injuries may imperil the circulation of the limb. At times a collateral circulation is established and recovery is complete. On other occasions Volkmann's ischæmic contracture supervenes. Sometimes the limb becomes gangrenous. Under what circumstances is arterial injury followed by recovery, ischæmic contracture or gangrene?

Recovery, ischæmic contracture or gangrene?—The vascular demands of the tissues of a limb are not uniform. The greatest susceptibility to ischæmia is shown by sensory and muscle nerve endings which lose their power of conduction within fifteen or thirty minutes. For this reason loss of the blood supply of a limb causes paralysis and anæsthesia even when the nerves are uninjured. The next most susceptible tissue is muscle which normally has a blood supply more vigorous than that of bone skin or ligament. If ischæmia persists for six or eight hours muscle dies whereas skin can survive as long as twenty four hours. Not only does muscle suffer more quickly than other tissues but its powers of regeneration are more limited. Unlike bone where an avascular sequestrum may be revascularised and replaced by living bone a muscle sequestrum can never be replaced by living muscle but only by fibrous tissue which undergoes contraction. The fate of the tissues of a limb after vascular occlusion depends therefore upon the speed with which the circulation is restored either by reopening original channels or by the development of collateral channels and the extent to which the relative demands of tissues have been met in the interval. If a free and vigorous circulation is established within about six hours recovery is complete. If the occlusion lasts more than six hours and the collateral circulation is not fully adequate tissues with the highest vascular demands suffer most. Muscles undergo necrosis and Volkmann's ischæmic contracture supervenes. If the collateral circulation is totally inadequate no tissue survives and gangrene results.

The collateral circulation—The adequacy of the collateral circulation depends upon three factors: (i) the age and constitution of the patient (ii) the local pressure of extravasated blood of splints and plaster or of body weight (iii) the degree of reflex vasospasm arising from the damaged segment of artery.

Age and constitution—Maximal vasodilatation of blood vessels is possible in the young but with advancing years much of the resilience of the arterial tree is lost. The elasticity of vessels is reduced by degeneration and

calcification, and by the arteriosclerosis of diabetes, syphilis or alcoholism. Shock, collapse and hæmorrhage, by reducing blood volume and pressure, and causing general circulatory embarrassment, are of great importance. The patient's general condition often determines the onset of gangrene after vascular damage¹ and blood transfusion or oxygen therapy may be essential. Vasodilatation should be encouraged by hot drinks, by the application of heat to uninjured limbs (but not to the injured limb) and by alcohol. Smoking which causes powerful vasoconstriction should not be permitted.

Local pressure on collateral vessels—If the circulation of a limb is in doubt, it must not be enclosed in a complete plaster cast. Swelling of an injured limb, continuing after the application of an unpadded plaster, may obstruct even the normal circulation and cause gangrene. When the main artery has been damaged and survival depends upon a collateral circulation, it is still more important that every source of external pressure should be removed. The limb may be supported on a cradle splint, if there is a fracture, light traction may be employed, and a plaster slab is sometimes necessary. But every encircling bandage, strapping or plaster cast must be divided. Even the pressure of body weight will sometimes obliterate the field of collateral circulation. After ligation of the external iliac or common femoral arteries, survival of the limb depends upon collateral vessels developing in the gluteal region. If the patient lies on his back, constant pressure on the buttock obstructs the circulation; he should be nursed on his face. If an artery is injured in a wound which becomes infected, the pressure of inflammatory exudates and œdema may obstruct the collateral circulation. Wide drainage of the wound is essential, the deep fascia must be divided freely in order to permit inflammatory swelling of muscles without constriction, no layer of the wound should be sutured. Even extravasated blood may cause sufficient pressure on surrounding tissues to reduce or obstruct the blood flow through collateral vessels. For this reason gangrene occurs more often when arteries are perforated by missiles than when they are ligated by surgeons. Statistics of 1914-15² showed that surgical ligation of the subclavian artery never caused gangrene, whereas perforating wounds of the subclavian artery caused gangrene in 9 per cent of cases. Gangrene occurred in 35 per cent of popliteal artery wounds and only in 26 per cent of popliteal artery ligations.

Reflex vasospasm—The influence of reflex traumatic arterial spasm on the collateral circulation has been discussed in the opening pages of this chapter. Injury to the wall of an artery, or thrombosis within the artery, sets up reflex vasoconstriction which affects not only the vessel itself but all the arteries of the limb including those upon which the collateral circulation depends. The vasoconstriction must be relieved by novocaine injection of the ganglionated sympathetic chain, or of the brachial plexus or lumbar nerves, and if this is not successful, by exposure and mobilisation of the artery or resection of the injured segment.

"The limb on ice"³—A limb threatened with ischæmia and possible gangrene should not be heated, for this increases the oxygen demand, hastens the onset of gangrene and favours the growth of pathogenic organisms. It

¹ G. Makins. Gun I & W. units in the Blood Vessels. Bristol 1919.
² Ministry of the Great War (Medical Services). L. n. 1. n. 1922 H. 1.0.
³ Lancet, Dec. 13 1914 H. 734.

has been recommended recently that the limb should actually be frozen by packing with ice or immersion in ice water,¹ because refrigeration inhibits bacterial growth and slows the local metabolism. These advantages are offset, however, by reduced dissociation of oxygen from oxyhaemoglobin. When it is still hoped that the limb will survive a middle course is advisable, it is kept cool but not frozen. If gangrene has already set in, but for any reason amputation must be deferred, a tourniquet should be applied and refrigeration used. Pain rapidly ceases the foul odour disappears and the patient's general condition is improved. A frozen limb can be amputated with no other anaesthetic. refrigeration inhibits the nerve endings, prevents pain and minimises shock.

VOLKMANN'S ISCHAEMIC CONTRACTURE

Until very recently it was believed that Volkmann's ischaemic contracture was the result of venous obstruction due to tight splints, bandages or plaster used in the treatment of elbow fractures. The view that the complication was almost invariably due to faulty treatment led to the award of large sums in damages against medical men who were held responsible. The theory of venous obstruction was based on the work of Brooks who ligated the veins of the sartorius muscle in dogs and of Jepson who ligated the femoral veins.^{2,3} Recent operative experience makes it evident, however that the lesions they produced experimentally were not the lesions we now find in human subjects. Volkmann⁴ himself, writing sixty years ago, believed that the contracture was due to 'a continuous stoppage of the arterial blood' and this is now well established. It is known that whereas total arterial occlusion persisting for about twenty four hours causes gangrene occlusion for shorter periods, or less complete occlusion causes ischaemic contracture. It is known that any source of arterial occlusion may be responsible, at any level, in any limb. An unduly tight plaster may certainly be the cause, because a tight plaster may obstruct the arterial flow, but this is far from being the usual cause. In the majority of cases ischaemic contracture is due to contusion of an artery, traumatic arterial spasm, thrombosis or embolism, perforation or rupture, traumatic aneurism or arteriovenous fistula, or any arterial injury or sequel of an injury which reduces the blood supply of the muscles of a limb.

Most recorded cases have been in the upper limb and have involved the brachial artery at the level of a supracondylar fracture of the humerus. Backward displacement of the small fragment stretches the artery across the fracture site causing contusion, laceration or complete severance. In 30 per cent of recorded cases the radial or ulnar arteries have been injured by fractures of the shafts of one or both forearm bones (Figs 224-225). Other fractures of the upper limb have been responsible in a few cases. Fig 236 is the arteriogram in a fracture of the neck of the humerus with ischaemic contracture due to injury of the axillary artery. In a fracture of the clavicle which I treated in 1930 a short inner fragment was forced directly backwards and lay at right angles to the skin occluding the subclavian artery,

¹ F. M. Allen, *Amer Jour Surg*, 1941 11 223.
² J. B. Murphy, *Jour Amer Med Assoc* 1914
 Incl 1219.
³ B. Brooks, *Arch of Surg* 1912 1 182.

⁴ P. N. Jepson, *Ann of Surg* 1926 lxxvii, 783.
⁵ D. S. Madlbeck, *Eur Jour Surg* 1931 cxlii 188.
⁶ R. von Volkmann, *Central f Chir* 1871 11 1881.
 (reprints f *Jour Internat Chir* 1874 iii 77).

the limb was pulseless and cold and although the circulation was improved by replacement of the clavicular fragment ischaemic contracture supervened. Fractures near the wrist may damage the radial artery and produce reflex vasospasm involving the whole length of the vessel throughout the forearm and even the brachial artery to the axilla thus causing ischaemic contracture.



FIG. 30

Fracture neck of humerus with ischaemic contracture

An arteriogram shows occlusion of the axillary artery at the level where it was struck by the lower fragment of the humerus at the time of its placement of the fracture. The arterial changes caused Volkmann's contracture. (*Report of Mr. J. C. H. H. Sep 131*)

of muscles proximal to the level of arterial injury. I have knowledge of two cases of ischaemic contracture of the forearm after osteotomies of the lower end of the radius for malunited Colles' fracture. In the lower limb cases have been reported after fracture of the upper shaft of the tibia, fracture of the neck of the fibula, traumatic rupture of the popliteal artery, perforation of the popliteal artery (Fig. 31) and embolism of the femoral artery. A child aged seven with bilateral compound fractures of the shafts of the

femora treated in an unpadded double plaster spica which was applied within one hour of injury, developed bilateral ischaemic contracture of the lower limbs the complication in this case being due to the tight plaster

Morbid anatomy—Muscles—The effect of depriving a muscle of blood is to cause necrosis and the formation of a muscle sequestrum^{1 2} Exactly as in the case of a bone sequestrum, microscopic examination shows that the general contour is preserved, but the tissue is dead individual muscle fibres can be recognised but there are no living nuclei either in muscle fibres or in interfibrillary tissue (Fig 238) Interfibrillary fibrosis such as occurs after denervation or infection of a muscle is never seen in ischaemic contracture, the tissue is inactive it is dead Surrounding the necrotic



FIG 237



FIG 238

Normal muscle

Muscle sequestrum in ischaemic contracture

Section of muscle after ischaemic contracture showing a typical muscle sequestrum The muscle fibres show normal contour but they are dead there are no nuclei There is fibroblastic cellular activity surrounding the sequestrum but no interfibrillary fibrosis such as occurs in denervated or infected muscle (By courtesy of Mr J M Barrie see p 133)

areas there is phagocytic and fibroblastic cellular activity Fibrosis eventually proceeds from without inwards and replaces the dead muscle with fibrous tissue which contracts and shortens **Nerves**—The same injury which bruises or tears the brachial artery may also bruise or tear the median, ulnar or musculo spiral nerves Even when there is no gross damage to nerve trunks, the limb may be paralysed by ischaemia of the nerve endings and in severe cases by actual degeneration of the nerve trunks due to ischaemia³ Many cases of Volkmann's contracture are therefore associated with nerve paralysis When the nerve lesion is due to ischaemia alone it usually recovers spontaneously, first in the posterior interosseous and later in the median and ulnar nerves

Clinical features of established contracture (upper limb)—Shortening of the forearm muscles causes typical deformity, with extension or hyper-extension contracture of the metacarpo phalangeal joints and flexion

¹ W R Bristow and Greenwood *Brit Jour Surg* 1933 3 473
² Crimmins *Volkmann's Ischaemic Contracture* *Brit Jour Surg* 1940 xxviii 244
³ J Laveuf *Contusion de l'Artere Humerale* *Jour de Chir* 1933 II 144

contracture of the interphalangeal joints¹ (Fig 239). In mild cases the contracture may be localised to one or two fingers. The degree of flexion of the fingers is increased when the wrist is dorsiflexed and diminished when



FIG 239

Typical deformity in Volkmann's ischaemic contracture (due in this case to brachial artery contusion in a supracondylar fracture of the humerus)



FIG 240

the wrist is flexed (thus distinguishing it from flexion deformity due to capsular injury in the finger itself). This is the basis of the technique of correction described by Robert Jones with the wrist flexed straight spatula splints are applied to each finger and a full length splint is then applied



FIG 241

A severe case of Volkmann's ischaemic contracture with posterior interosseous medial and ulnar paralysis and permanent crippling despite many operations and years of treatment

to the forearm and hand and the wrist is gradually dorsiflexed. A still better method of correction is to support the wrist in a splint in a very slightly dorsiflexed position and apply elastic traction loops to each individual finger. Treatment must be prolonged improvement in the degree of

¹ It is to be recognized that the flexion of the fingers at the interphalangeal joints is not a power of the interphalangeal joint itself, but is a result of the contraction of the extensor muscles of the forearm at the interphalangeal joint. The interphalangeal joints which have a very short flexor digitorum profundus and a very long extensor digitorum profundus are not able to extend the fingers at the interphalangeal joint.

contracture may be expected to continue for at least two years. Residual deformity may then be treated by a muscle slide of the forearm flexors from the internal epicondyle, by lengthening the tendons in the lower forearm, by shortening the forearm bones or by arthrodesis of the wrist. Despite all these measures a really severe Volkmann's contracture causes permanent crippling. Main en griffe and ape thumb deformities due to ulnar and median paralysis may be superimposed on flexion contracture due to fibrosis of the forearm muscles. Fig. 241 which represents the result of many operations and years of treatment in a particularly severe case is the best possible illustration of the importance of preventive treatment.

Early clinical diagnosis—If disaster is to be avoided the diagnosis must be made within a few hours. The one clinical sign of importance is absence of the radial pulse. The fingers are usually cold, slightly swollen and either cyanosed or pale. There is sometimes burning pain in the forearm and hand but the onset is often painless. Insensitivity of the fingers and immobility in a slightly flexed position are later signs. If there is actual flexion contracture the diagnosis has been made too late; the damage has been done.

Preventive treatment—Absence of the radial pulse in fractures of the elbow or forearm must be regarded with grave suspicion. If there is the slightest sign of circulatory embarrassment the following steps are of immediate urgency:—

- 1 Give a brachial plexus block anæsthetic with 2 per cent novocaine. Not only is this a suitable anæsthetic for subsequent procedures but it is a therapeutic measure which may avert the catastrophe.
- 2 If there is an elbow fracture still un-reduced reduce it at once by manipulation. Reduction of the fracture will often relieve the vascular complication (Fig. 242).
- 3 Exclude the possibility of external pressure by cutting through the whole length of any plaster strapping or bandage which encircle the limb. It is usually inadvisable to remove the whole plaster, but remove at least the front half.
- 4 If the elbow is swollen and has been immobilised in flexion reduce the angle of flexion in order to prevent pressure in the antecubital space. If necessary extend the elbow beyond the right angle and maintain immobility and reduction of the fracture by a posterior plaster slab.
- 5 If despite these precautions the pulse remains absent and circulation is obviously impaired do not wait more than an hour or two. Expose the artery at the level of injury. Carefully avoid damage to all branches. Raise the vessel from its bed, clean it of areolar tissue

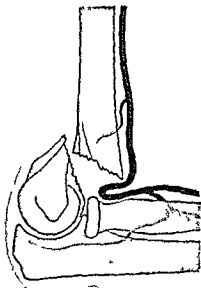


FIG. 242

Flexion of a swollen elbow compresses the brachial artery, especially if there is an unreduced supracondylar fracture.

and apply warm saline douches. Gently massage the vessel. If the pulse does not return, inject papaverine ($\frac{1}{4}$ gr) intravenously. If the vessel is bruised, contused or lacerated, apply ligatures above and below the lesion and resect the injured segment.

Ischaemic contracture in the lower limb—Injury to the popliteal and femoral arteries usually causes gangrene rather than ischaemic contracture (p. 127). Nevertheless ischaemic contracture does occur in the lower limb



FIG. 243

Ischaemic contracture of the toes is more common than is generally realised (see text). For correction the metatarso-phalangeal joints are held flexed while elastic traction to a hinged plaster flap extends the interphalangeal joints. Flexion exercises are practised against the resistance of the elastic band.

and in gunshot wounds and infected fractures which have been treated in tight plaster. This complication is far more common than is generally recognised. Arterial injury is not the only cause of ischaemia; tight plasters can not be exonerated. In the last two years I have seen many infected compound fractures of the tibia which had been treated from an early stage in plasters fitting so closely that circulation of the toes was impaired. I have often seen loss of active movement of the toes even when there was no nerve injury; the toes become flexed and contracted; the deformity is resistant and despite good union of the fracture incapacity is prolonged. I firmly believe that this complication is due to obstruction of the arterial flow by a tight unpadded plaster causing ischaemic contracture of the muscles.

The complication is to be avoided by greater care in the use of the unpadded plaster cast and by the more general use of elevation of the limb for several days or weeks after wounding in order that swelling of the limb shall be minimised. An unpadded plaster should not be applied immediately after wounding, after operative reduction of a fracture, after sequestrectomy or after any other treatment of an infected wound which will be followed by

reaction. In all these cases a thin wool bandage should be smoothly applied and the use of a non-padded cast deferred for several weeks. The circulation of the toes and the power of active mobility must be watched with care. At the first sign of pallor, cyanosis or immobility the plaster must be cut throughout its length; if necessary it must be divided. In late cases where flexion contracture of the toes has already developed it should be corrected by elastic traction (Fig. 243) and in severe cases by manipulation or tenotomy.

GANGRENE DUE TO FRACTURES

Unpadded plaster casts—The application of an unpadded plaster cast to a fractured limb within a short time of injury and before reactionary swelling has occurred is dangerous. Pressure within the rigid cast may become so great as to obstruct the arterial flow and cause not only ischæmic contracture but even gangrene. At this early stage, plasters should be lightly padded with wool bandage the unpadded cast being applied only after several weeks. Whether the plaster is padded or unpadded, the circulation of fingers and toes must be watched carefully and tested frequently. It is not enough to apply digital pressure and confirm that the anæmic area refills with blood when pressure is withdrawn, this may be observed even after complete circulatory stasis, because some blood remains in the digit. The surgeon must be satisfied that the return of blood is brisk, that the digit is warm, that it is pink and that it is neither cyanosed nor pallid. Unless he is satisfied the plaster must be bivalved *at once* and the front half removed. If the circulation does not return, and the tibial (or radial) pulse cannot be felt the diagnosis of arterial injury must be accepted and the necessary steps be taken immediately (p. 125).

Contusion of arteries by fracture—The types of arterial injury which may result from impact of the fragments of a fractured bone have already been discussed. They include concussion of the artery with traumatic spasm, contusion with spasm, contusion with thrombosis and spasm, embolism with spasm and perforation or rupture with hæmatoma, aneurism or arterio-venous fistula. Of these injuries the most common is simple contusion with reflex vasospasm. In the upper limb the radial and ulnar arteries may be contused by the forearm bones, the brachial artery by the lower end of the humerus the axillary artery by the neck of the humerus and the subclavian artery by the clavicle. The potential collateral circulation in the upper limb is good, and although reflex vasospasm may cause ischæmic contracture there is seldom complete and persistent arrest of the circulation causing gangrene. In the lower limb the risk of gangrene is twice as great.¹ The femoral artery is in danger where it is anchored close to the femur by the fibrous arch of the adductor magnus. Still greater is the danger of the popliteal artery in the lower part of the popliteal space.

Gangrene after high oblique fracture of the tibia and fibula—The popliteal artery at its bifurcation lies close to the tibia and in actual contact with the fibula the neck of this bone being grooved by the anterior tibial branch. The vessel is anchored by the fibrous arch of the soleus, and by the passage of the anterior tibial branch over the interosseus membrane. If there is a fracture of the adjacent bones the vessel cannot easily escape contusion or traction injury. The bone injury which is nearly always responsible is a high fracture of both leg bones, oblique from before, upwards and backwards, the main shaft fragment is displaced upwards and backwards and the artery is impaled upon its sharp margin (Fig. 244). Of seven fractures which I have seen of this type and with this displacement, no less than five were

¹ H. Dodd. Gangrene following Fractures. *Ind Jour Surg*, 1914, xxii, 230

complicated by gangrene of the foot and leg¹ (Fig 245) This frequency is the more striking when it is recalled that after searching the literature of the world Dodd could find only one case in twenty years (1914-34) It may be that cases were not then recognised or reported, but the greater use of the unpadded plaster cast in recent years cannot be ignored The collateral circulation after injury to the popliteal artery, at a level below the anastomoses of the knee, depends upon small and insignificant vessels The



FIG 244



FIG 245

Gangrene due to high oblique fracture of the tibia

From the point of view of vascular complication this is the most dangerous of all fractures Five out of seven fractures of this type were complicated by gangrene The main fragment of the tibia is displaced upwards and backwards and its sharp upper margin strikes the popliteal artery at its bifurcation, a level where it is anchored and cannot escape Anticipate the danger Defer the application of plaster for three days If the tibial pulse cannot be felt block the sympathetic with novocaine Prepare to expose the artery

pressure of plaster, even if carefully applied, may well hinder the development of so feeble a circulation

High fracture of the shafts of tibia and fibula must therefore be recognised as a dangerous injury The possibility of arterial damage must be anticipated and the application of plaster be withheld for several days The position of the fragments can be controlled, and redisplacement prevented by skeletal traction from a pin in the lower shaft of the tibia the limb being supported

¹ One other case I saw a direct bearing I operated on a nurse aged twenty-eight for severe gangrene due to arrested growth in adolescence of the lower part of the tibia (epiphysis) The bone was almost but not quite divided with an osteotomy the fracture was exposed manually the distal fragment corrected and a padded plaster applied The circulation of the toes was believed by the resident surgical officer to be satisfactory until the third day when they were obviously pulseless Exploration of the popliteal artery showed no bruising or laceration but it was strongly contracted at the level of the fibula arch of the soleus Extensive amputation at a straggling distance restored the circulation and three days later a gangrenous limb was amputated Post mortem examination confirmed that the arterial lesion had been a simple traumatic vasospasm with no gross injury or thrombosis The operation if performed earlier might well have been successful but the diagnosis was made too late

in a Thomas splint. The tibial pulse and the circulation of the toes must be watched with great care. At the first sign of circulatory failure a spinal anæsthetic should be given (or a paravertebral novocaine injection) and the popliteal artery exposed at its bifurcation by splitting the upper fibres of the soleus. If stripping the vessel removing the areolar tissue and injecting papaverine do not restore the circulation arteriotomy must be considered particularly when there is actual bruising or thrombosis of the vessel.

In the treatment of other injuries of the leg the surgeon must remember the susceptibility of the popliteal artery and its branches. Contusion of the anterior tibial artery against the neck of the fibula has caused gangrene from bumper injuries even when there was no fracture. It is obvious therefore that the technique of reducing fractures of the tibial tuberosity by tightly encircling the fragments with a tourniquet and hammering them with a sandbag is to be regarded with grave suspicion. One case has been recorded where this treatment was complicated by gangrene. There must also be care in the reduction of tibial shaft fractures by traction apparatus where counterpressure is applied in the popliteal space.

GANGRENE DUE TO TOURNIQUETS

More limbs have been lost by the use of tourniquets than have been saved. The correct first aid treatment of hæmorrhage from a wound is often to keep the patient at rest and do no more. Alternatively the treatment is to maintain pressure over a large pad applied to the wound itself. In teaching first aid and ambulance workers this treatment is usually dismissed in a few words, hours are then devoted to the consideration of pressure points and the application of tourniquets. It is to be expected therefore and it is in fact the case that when in ambulance man sees a wound he at once thinks of a tourniquet. If a tourniquet is not applied tightly enough the veins are obstructed but not the arteries. I have vivid recollection of a child who for this reason almost bled to death, hæmorrhage from the wound ceased as soon as the tourniquet was released. The tourniquet had been far more perilous than the wound. On the other hand if the unfortunate ambulance worker applies the tourniquet too tightly he is more than likely to cause gangrene. It is not enough to mark the patient's forehead with a T and remove the tourniquet at intervals because if traumatic arterial spasm is once induced it continues whether the tourniquet is removed or not. Vaso-spasm, ischæmic contracture and gangrene due to the application of tourniquets have now been recorded by Esmarch,¹ Wallis,² Ducasting,³ Griffiths,⁴ Trueta,⁵ Barnes and Cohen.⁶ The danger arises not only from the length of time that the tourniquet is in position but from the force with which it is applied. Gangrene necessitating amputation of the arm has been due to an Esmarch rubber tourniquet removed within forty five minutes (Fig. 246). The upper limb is certainly more dangerous than the lower limb but in neither upper nor lower limb is there complete safety except with the controlled pressure of a pneumatic tourniquet. The partly skilled first aid worker who has been taught to improvise tourniquets from bandage or cord tightened with pieces of wood is a most dangerous person. There is danger in applying

¹ F. Arch (18) J. B. C. Jo. r. Surg. 1910 xxx 5

² I. Wall. Practitioner 1911 ix 4 9

³ R. Ducasting. B. U. Soc. Med. Hosp. 1919 xl 604

⁴ D. Griffiths. Brit. Jour. Surg. 1910 xxvii 1

⁵ Trueta and Barr. J. t. Jo. r. 1911 xxx 1

⁶ S. W. C. J. t. Hosp. Rep. 1911 xc 2

the tourniquet too tightly there is even greater danger in not applying it tightly enough. The only danger which is remote is that lack of a tourniquet will permit fatal hæmorrhage. It has been known for two hundred years that completely severed arteries usually cease bleeding within a few minutes.



FIG. 46

Gangrene due to a tourniquet

A first operation was performed with a flat Esmarch rubber tourniquet on the upper arm. It was removed in forty five minutes. Persistent traumatic arterial spasm caused gangrene. Be rare tourniquets. Use only the pneumatic type.

(p 108) It is not surprising therefore that surgeons in the Spanish war strongly condemned the use of tourniquets. It is not surprising that a Committee of the British Medical Research Council recently voted against the inclusion of tourniquets in life boat equipment. First aid workers should use local pressure on the wound. Surgeons should use pneumatic tourniquets.

IMMERSION FOOT SHELTER FOOT, TRENCH FOOT

A pilot crashed in the North Sea. For fourteen days and fourteen nights he floated in a rubber dinghy awash with the waves cold wet benumbed. He had no food and on the eighth day he drank his last ration of water. He could not have survived but for an incredible chance. On the twelfth morning a wary sea gull seeking refuge hovered and landed on the edge of the perilous craft. The airman showed restraint he waited and when the bird raised its wing he clutched and caught it. With relish and no aversion he ate the bird he sucked its brains and enjoyed its blood he ate a small fish in its belly. On the fourteenth night in the moonlight he saw a motor torpedo boat. He stood on his feet waved and shouted and was taken aboard in great spirit. He stood on his feet but they were gangrenous (Fig. 24.) Five hundred hours continuous exposure to cold and wet had caused immersion feet¹ a peril to which armen are exposed in dinghies sailors in life boats soldiers in trenches and bombed civilians in dug-outs (trench foot² and shelter foot³). The circulatory failure is allied to frost bite (Fig. 248) and is due to arterial spasm induced by cold. Contributory

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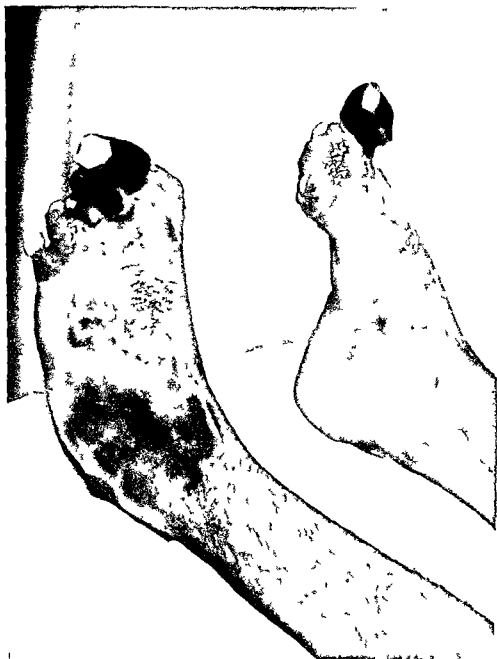


FIG 247

Immersion feet

Exposure to cold causes reflex vasospasm and sometimes thrombosis of the vessels with gangrene of superficial tissue and toes. This patient was a pilot adrift in a dinghy for fourteen days. He was without water for six days. His life was saved by a sea gull.

(See text. Treatment supervised by Air Commodore G. Keynes and plastic surgical treatment by Mr A. McIndoe.)

factors are damp, venous congestion or thrombosis which aggravate the arterial spasm, and exposure, hunger or debility which lower the general circulation. The feet become cyanosed and blue. In mild cases the circulation may recover, but in more severe cases gangrene develops in the toes and in the tissues over the heels and malleoli. The feet at first look worse than



FIG 248

Ulceration following frost-bite

Whereas immersion foot is due to vasospasm frost bite is due to actual freezing with the formation of minute crystals of ice in the skin^{1,2}

they are, and if conservative treatment is adopted it will often be found that the gangrene is only skin deep and that deeper structures are still alive. Persistent vasospasm in severe cases may cause thrombosis of the tibial arteries. In these patients even if gangrene has been avoided acutely painful vasoconstrictive attacks may persist over a period of months or years. Leriche reports such crises in cases of trench foot, with complete relief from pain and improvement in the circulation, after resection of the thrombosed vessels.³

CRUSH SYNDROME—"TRAUMATIC ŒDEMA"

The first accounts in English literature of the crush syndrome were published in 1941 at a time of intensive bombing, when many victims were buried for hours or days under fallen masonry and debris. The same clinical condition was recorded after the Messina earthquake as "acute pressure necrosis" and although unrecognised in this country, it must have occurred in mine accidents and after other injuries where there is sustained crushing of the muscles of a limb.

Clinical features—As a rule there is no fracture, external wound or hæmorrhage, but only abrasion of the skin and discomfort on using the muscles which have been crushed. After a latent period which may last several hours the blood pressure falls to severe shock levels. This is due to plasma loss into the damaged area, and is accompanied by hæmoconcentration (up to 160 per cent Hb) and œdema of the limb. Sometimes the circulation becomes impaired. Arterial pulsation is diminished or even lost and there

¹ Frost-bite. In *Immersion Foot*. (Guide to the Preservation of Life at Sea after Shipwreck. Med. at Naval Coll. War Med. Coll. 1941.) H.M. Stationery Office, 1941.

² W. G. Bigelow. "A clinical description and treatment of frost-bite" (with a survey of literature). *Canad. Med. Ass. Jour.* 1942, Dec. 2, 1111-19.

³ R. Leriche. *The Surgery of Vain*. (translated by Arcliball Young. London 1939) 479.

are signs of incipient gangrene. It seems probable that when there is circulatory failure it is due to injury of arteries sustained at the time that the muscles were crushed in the same way that paralysis may occur from simultaneous compression of the peripheral nerves. Certainly gangrene is not an essential feature of the syndrome, and the renal changes described below occur as a direct result of muscle crushing independently of the circulation of the limb, and no matter whether the limb at this late stage is amputated or not.¹

Renal changes—The urinary output is low from the beginning. After a few days the output is progressively diminished and there are signs of renal failure. There is acidosis and myohæmoglobinuria. The blood urea rises to 300 or 400 mg per cent and death from uræmia occurred in 60 per cent of the sixty cases of which we have detailed information.² Post mortem examination shows that the renal failure is due to necrosis of the distal convoluted tubules. The appearances are similar to those seen in the kidneys of patients dying from mismatched blood transfusions, where a hæmoglobin pigment rendered insoluble by high concentration and an acid reaction is precipitated in the tubules (probably as acid hæmatin). In the crush syndrome the pigment is not hæmoglobin but myohæmoglobin, and it comes from the damaged muscle of the limb. Mechanical obstruction of the renal tubules by this pigment together possibly with toxic agents also released from the necrotic tissue in the crushed limb accounts for the kidney changes and renal failure.

Treatment—Renal failure must be guarded against by alkalinisation and the administration of fluids. An alkaline mixture containing 30 gr sodium citrate 15 gr sodium bicarbonate ad 1 oz aqua is given at once if possible even before the patient is released up to a total of 120 gr citrate. It is continued thereafter at the rate of 500 gr daily. If the patient is not seen until a later stage and the urine must be alkalinised rapidly, 3 per cent sodium citrate is given intravenously. The fluid intake must be at least 3 litres daily.

Plasma leakage or "shock"—The leakage of plasma into the injured area must be treated by serum or plasma infusions in the "pre shock" stage (while there is hæmoconcentration but before the blood pressure falls see p 206).

Local treatment—The injured limb should be elevated. The body as a whole may be warmed but the limb should be kept cool. The theoretical suggestion that early amputation is advisable finds little support but circulatory failure may call for paravertebral novocaine injection or exposure of the artery.

¹ G Bywaters. Crush Injuries (with ref.) *Brit Med Jour* 1941 ii 29.

² Medical Research Council Sub Committee on Traumatic Shock 1942.

AUTHOR'S NOTE

I am glad to record my indebtedness to Mr S. M. Cohen of London not only for the illustrations he has allowed me to use (Figs 229, 232, 239, 240 and 246) but even more for the stimulus of his infectious enthusiasm in clinical investigation and postgraduate teaching. I am also grateful to Professor H. J. Seddon and the team at Oxford whose research he has inspired notably Mr N. Bremner Hight who entrusted me with original material even before publishing it himself (Figs 224, 225 and 236) and Mr J. M. Barnes who gave me access to his valuable experimental work on *Ischemic Contracture* (Figs 237, 238). In this chapter I have referred to Mr D. H. Griffith's Hunterian lecture on Volkmann's Ischemic Contracture, this is a masterly contribution which I would commend to every reader.

CHAPTER VII

NERVE INJURIES COMPLICATING WOUNDS AND FRACTURES

Peripheral nerve injuries were formerly classified into two groups—*anatomical lesions* in which the nerve was completely divided and ‘*physiological lesions*’ in which despite an appearance of anatomical continuity there was loss of conduction of the nerve. This subdivision was not entirely satisfactory. An anatomical lesion was clearly recognisable and always associated with Wallerian degeneration in the distal nerve stump, but physiological lesions included different types of injury, some with Wallerian degeneration and some without. It is better to define three groups of nerve injury: (1) *complete division of the nerve*, (2) *nerve lesion in continuity*, and (3) *transient nerve block*. Complete division of a nerve causes a break not only in the axons but also in the supporting structures, epineurium, perineurium and endoneurium, and it is the lack of continuity of these structures which explains the difficulty of spontaneous regeneration. A lesion in continuity due to severe compression or crushing of a nerve again interrupts the axons and causes Wallerian degeneration, but the supporting structures remain in continuity, so that spontaneous recovery is the rule and regeneration occurs with greater speed and accuracy than after complete division. A transient nerve block due to contusion or ‘concussion’ of a nerve does not cause Wallerian degeneration, spontaneous recovery occurs more quickly than in any other type of lesion, and normal function is restored within a week or two. These three types of nerve injury can be described in the terms of a recent nomenclature as *neurotmesis*, *axonotmesis* and *neurapraxia*.¹

TYPES OF NERVE INJURY

Complete nerve division—neurotmesis—Histological examination of the nerve distal to the lesion shows degeneration of axis cylinders, fragmentation and absorption of myelin sheaths and proliferation of the neurolemmal cells of Schwann. The Schwann cells form columns which act as guiding tubes for the regenerating axons, at the cut surface of the nerve they grow out seeking to reach the proximal stump and to direct the sprouting axons across the gap.² The nerve proximal to the lesion shows retrograde degeneration for about 1 cm. Sprouting of the axons begins after an interval of about ten days and new buds then spread out in search of the columns of

¹ *Tmesis*—cutting or division as in tenotomy, hence neurotmesis implies division of the whole nerve, axonotmesis implies division of axons but not of the supporting structures of the nerve, neurapraxia implies action, hence neurapraxia implies transient loss of action or of conduction. These terms were suggested by Prof. H. Cohen and introduced by Prof. H. J. Seddon. “A Classification of Nerve Injuries.” *Brit. Med. Jour.* 1941, ii, 751.

² J. P. Young. “Nerve Regeneration—Importance of the Peripheral Stump.” *Lancet* 1940, ii, 128.

Schwann cells. If no contact is established between central and peripheral stumps the two nerve ends become expanded into globular masses—a large central neuroma and a smaller peripheral glioma. On the other hand if contact is established axon buds grow into Schwann cell tubes and the nerve fibre regenerates. The functional result is determined by the proportion of nerve fibres which regenerate successfully. When apposition is inaccurate many central axons fail to find a peripheral tube down which to grow. Spontaneous regeneration is therefore unusual after complete division. If the nerve ends are sutured in accurate apposition much better function is regained, provided that care is taken to avoid deep insertion of sutures which deflects the growth of axons into irregular whorls. Even when apposition is accurate and there is no obstacle at the suture line considerable criss crossing of fibres is inevitable. Central axons grow blindly down any available peripheral tube and if the nerve trunk is mixed motor axons may grow down sensory channels and sensory axons down motor channels. The functional result is therefore imperfect, particularly for example after division of the median and sciatic nerves. Regeneration in a simple nerve which includes only sensory fibres shows similar crossing in the inaccuracies of localisation which often remain permanently. In the musculo spiral nerve consisting largely of motor fibres the result is relatively good. Regeneration occurs at a rate variously estimated from 1 to 4 mm a day. It is possible to make a reasonably accurate estimate of the time of expected recovery by measuring the length of nerve involved allowing an average rate of 2 mm a day and adding ten days for crossing the suture line.

Lesion in continuity—axonotmesis—If a nerve is compressed or crushed but not severed and there is no loss of anatomical continuity axis cylinders undergo Wallerian degeneration as they do after complete division and proliferation of Schwann cells at the level of injury gives rise to a fusiform enlargement (neuroma or neuroglioma). But the gap between proximal and distal segments of the nerve is no more than a break in the axons. Regeneration occurs spontaneously and at a considerably faster rate than after complete division. Moreover the general architecture of the nerve is preserved at the level of injury and central axons grow into appropriate peripheral channels with minimal crossing of fibres so that good functional recovery occurs even in mixed nerves.

Transient block—neurapraxia—Simple contusion, concussion or traction may cause temporary loss of conduction of a nerve with no actual degeneration of axis cylinders in the distal segment. The lesion is often incomplete. Subjective alteration of sensation such as tingling or numbness may be the only disability. Sometimes there is complete motor loss with incomplete sensory loss. Recovery takes place within a week or two and often occurs with equal rapidity throughout the whole area of distribution.

Treatment of nerve lesions—Paralysis due to transient nerve block recovers spontaneously and no special treatment is needed, paralysis due to a lesion in continuity usually recovers spontaneously and exploration of the nerve is unnecessary, paralysis due to complete division seldom recovers spontaneously and suture of the nerve is essential. The differentiation between these lesions cannot be established in early days with any certainty, and exploration is sometimes advisable in order to determine the type of injury. The probabilities can be summarised as follows —

- 1 If there is an open wound and paralysis is complete, the nerve is probably divided and nerve suture is necessary
- 2 If there is an open wound and paralysis is incomplete, the symptoms may be due to a lesion in continuity or transient block which will recover without operation, but they may also be due to complete division of part of the nerve, which necessitates suture Unless recovery is prompt, exploration is advisable
- 3 If there is a closed fracture of the type often associated with nerve concussion contusion or traction (*e.g.*, ulnar paralysis in elbow injuries) the paralysis is probably due to transient block and no operation is necessary
- 4 If there is a closed fracture of the type often associated with severe nerve contusion or laceration (*e.g.*, musculospiral paralysis in fractures of the humerus or median paralysis in supracondylar fractures) the nerve injury is probably a lesion in continuity, but it may be a complete division and unless there are signs of recovery within three or four months exploration is advisable

The optimum time for exploration of a nerve is determined primarily by the importance of avoiding infection. The operation must not be performed until it is reasonably certain that the wound will heal by first intention. Within limits the interval between the wound and the operation has no influence on the time taken for recovery.¹ There is certainly no harm in waiting six months but it must be recognised that the longer operation is delayed the more serious are the changes in the paralysed limb.² Moreover, if the interval is greater than eighteen months the chances of successful recovery are slight.³

NERVE INJURIES IN WOUNDS

Recent wounds—Divided nerves should not be sutured at the time of the original operation of wound excision. The surgeon can seldom be sure that a wound will heal by first intention, and if there is the slightest risk of infection there can be no justification for nerve suture. The primary operation should be concentrated on the task of minimising infection and securing the most rapid possible healing. If the wound heals without infection the nerve can be explored within two or three weeks, otherwise the nerve suture is deferred until about two months after healing is sound. If the wound is associated with a compound fracture, suture of the nerve is further delayed until the fracture is united and neighbouring joints are mobilised.

Exploration of nerve and neurolysis—As a rule it is better to use a local rather than a general anaesthetic. A generous incision is made and the nerve is identified above and below the injury before any attempt is made to trace it through scar tissue at the level of injury. If the nerve is found to be completely divided with a well defined proximal neuroma and distal ghoma there can be one decision only⁴, the nerve bulbs must be excised

¹ Med. and Research Council Committee on Injuries of the Nervous System, 1920. Special Report M.R.C. No. 1.

² H. Cairns and J. J. Young. Gunshot Wounds of the Upper Limbs. *Lancet* 1941, II, 123.

³ H. Platt. *Surgery of Peripheral Nerve Injuries of Warfare*. Bristol 1941.

⁴ "Build suture."—The exception to this observation that there is one decision and one only arises in a case where despite full mobilisation of the nerve and fixation of neighbouring joints the gap to be bridged will be too great for immediate suture without tension. Bulb suture is then a valuable alternative being followed together by full flexion of the joints and secured by a heavy stitch through the scar tissue. (a) Those at gradual stretching after this preliminary operation may give sufficient length to permit excision of the bulbs and formal suture at a later date.

and the nerve sutured. If however there is anatomical continuity and a fusiform enlargement the decision must be made whether it is a lesion in continuity not calling for excision and suture or a complete division in which proximal and distal bulbs are joined by scar tissue into a single fusiform spindle. If the central part of the expansion is hard dense and almost

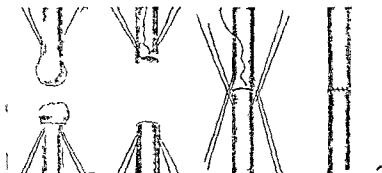


FIG. 943

Operative suture after complete division of nerve. The nerve bulb is excised. Guide sutures are used for orientation and approximation. The finest thread or silk stitches are inserted through the nerve sheath.

cartilaginous the division is very probably complete. This may be confirmed by electrical stimulation. If the lesion is complete a proximal stimulus will fail to cause contraction of muscles innervated from a lower level; a distal stimulus will not give rise to any sensation of tingling in the area supplied by the nerve. If the surgeon is in doubt it is better to be satisfied with the neurolysis already completed than to make the error of excising a regenerating nerve. The guiding principle should be radical nerve exploration—conservative nerve operation.¹

Suture of nerve.—The nerve ends must be mobilised with such a degree of freedom that after the nerve bulbs have been excised there is easy contact without tension. It is usually necessary to expose a considerable length of nerve to flex the joints above and below and sometimes to gain length by transposing the nerve to a new bed (for example transposing the ulnar nerve to the front of the elbow joint). The proximal neuroma and distal glioma must be cut back with a safety razor blade or sharp scalpel until the whole area of intraneural scarring has been removed, the typical faggot-like mass of nerve fibres being exposed and the nerve sheath showing normal retractility. The nerve ends are then correctly orientated and guide sutures are inserted by which to hold them in accurate apposition. Fine linen thread or fine silk (No. 0 Deknatel) are better suture materials than non-chromicised 000 000 catgut. The sutures must include the nerve sheath only and not be placed so deeply as to disturb the underlying axis cylinders. There must be no crowding or compression of the fibres; interrupted sutures are therefore better than a continuous stitch. The fibres must just touch; if there is to be any defect at all it should be a slight gap rather than a crowding. Every fibre must be enclosed within the sheath so that it is in apposition with an opposite fibre. The repaired nerve is then replaced in a muscular bed free from scar tissue. The flexed position of the joints by which apposition of the

nerve ends and freedom from tension was secured must be maintained during suture of the wound, and for two or three weeks after operation, by means of a light plaster cast

"Fibrin suture" of nerves—Suture material, no matter how fine or how carefully inserted, is a mechanical obstacle which tends to interrupt the line of growth of budding axons and deflect them from their course. A method has been devised of holding nerve ends together with a "glue" of concentrated blood plasma, coagulated by the addition of chicken embryo tissue extract.¹ Within about two minutes of adding the extract, the plasma clots to a jelly. This holds the nerve ends in apposition and allows axons to bridge the gap with greater accuracy and speed than they can cross a scar sutured with thread or catgut.

For further details of the treatment of nerve injuries in wounds the reader is referred to Prof. H. J. Seddon's excellent article in *Surgery of Modern Warfare*.² The reader should also consult *Aids to the Investigation of Peripheral Nerve Injuries*, Medical Research Council War Memorandum No. 7, 1942, H.M. Stationery Office.

NERVE INJURIES IN CLOSED FRACTURES AND DISLOCATIONS

Nerve injuries which complicate fractures and dislocations may be classified chronologically into three groups: (1) primary nerve injuries sustained at the time of bone injury by ischaemia, compression, contusion, traction or laceration of the nerve; (2) secondary nerve injuries developing after some weeks or months as the result of friction or late compression; (3) delayed nerve injuries developing after many years from stretching of the nerve by gradually developing deformity.

1. Fractures with Primary Nerve Injuries

Ischaemic nerve lesions—These have already been considered under Volkmann's ischaemic contracture (p. 121). Loss of blood supply for thirty minutes causes temporary paralysis of the sensory and motor nerve endings. Ischaemia for a longer period may cause Wallerian degeneration but it is a lesion in continuity which recovers spontaneously within a few months.

Compression nerve lesions—Reference has been made to compression injuries of nerves accompanying crush oedema in air raid victims whose limbs are crushed for many hours by fallen masonry (crush syndrome, p. 132). Paralysis may occur even when the more serious crush syndrome is avoided. The external popliteal, sciatic, musculospiral or ulnar nerves are usually involved, the nerve being compressed between the adjacent bone and the fallen weight. The injury is a lesion in continuity (axonotmesis) and recovery is spontaneous.

Contusion nerve lesions—A nerve may be contused by a dislocated or fractured bone. When the carpal lunate bone is dislocated forwards it is forced into the confined space beneath the anterior annular ligament, and the median nerve is often injured by the blow.³ (Fig. 250). The paralysis is sometimes incomplete and due to transient block, but even when injury is more severe it is a lesion in continuity which recovers fully. In about 15

¹ J. J. Young and P. B. Medsger, "Fibrin Suture of Peripheral Nerves," *Lancet*, 1940, II, 176.
² Seddon, *Surgery of Modern Warfare*, vol. II, 5th edition, H. Bailey, J. & A. Livingstone, Edinburgh.
³ Watson Jones, "Carpal Semilunar Dislocation with Nerve Lesion," *Lancet*, 1903, I, 1071.

per cent of supracondylar fractures the median or ulnar nerves are contused by the lower margin of the main fragment of the humerus¹ (Fig 251) As a rule recovery is complete within a few weeks (neurapraxia) or at the most within about three months (axonotmesis) and no treatment is required other than reduction of the fracture. Occasionally however the nerve is completely severed even in closed fractures.

Traction nerve lesions—Traction nerve lesions causing transient nerve block are very common in fractures and dislocations particularly of the elbow joint where the ulnar nerve is involved and the shoulder joint where the brachial plexus is injured. More severe traction injury causing a lesion in continuity (axonotmesis) sometimes occurs in shoulder dislocations and in injuries of the knee which stretch the lateral popliteal nerve. Traction causing complete division of nerves seldom occurs except in suprascapular injuries of the brachial plexus.

Ulnar paralysis—The ulnar nerve is held in the post condylar groove by a roof of fibrous tissue and a thin mesentery. It is fixed to the forearm by its muscular branches. If the elbow joint is forced into cubitus valgus the nerve is stretched. Valgus deformity of the elbow often occurs as the result of a fall on the outstretched hand which may cause avulsion of the epiphysis of the internal epicondyle outward subluxation or dislocation of the elbow or a supracondylar fracture with outward displacement of the lower fragment (Figs 252-256). In these injuries all the structures on the inner side of the joint are stretched and ulnar paralysis is frequent.^{2,3}

Similarly if the forearm bones are displaced forwards the nerve is stretched over the back of the lower end of the humerus. Supracondylar fractures with forward displacement and forward dislocation of the elbow with fracture of the olecranon are unusual injuries but when they occur there is a high incidence of ulnar paralysis. On the other hand if there is backward displacement of the forearm bones the nerve is displaced



FIG 250

Dislocated lunette bone with median paralysis from contusion of the nerve

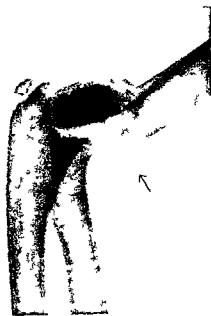


FIG 251

Supracondylar fracture of humerus with median paralysis due to nerve contusion

W. R. Britton, Complications of Supracondylar Fractures. *J. J. Orth. Surg.* 1934, 1: 1. 4.
¹ Hatt, The Ulterior Nerve Complications of Fractures and Dislocations of the Elbow. *Robert Jones Orth. Soc. Vol. 6, Oxford Univ. Press, 1924, 19.*
² Watson Jones, Fractures of the Nerve Lesions in Injuries of the Elbow. *Am. J. Orth. Surg.* 1930, 12: 1.

backwards from its groove it gains free mobility and escapes traction injury. Ulnar palsy does not therefore occur in elbow dislocations and



FIG 202



FIG 203



FIG 204



FIG 205



FIG 206

Injuries of the elbow with ulnar paralysis due to traction injury

Fig 202 avulsion of epiphysis of internal epicondyle Fig 203 subluxation of elbow with inclusion of epicondyle on inner side Fig 204 outward dislocation of elbow Fig 205 supracondylar fracture with outward displacement Fig 206 supracondylar fracture with forward displacement

fractures with backward or with backward and outward displacement. Like contusion nerve injuries these traction injuries are often incomplete and the lesion is no more than a temporary loss of conductivity. Some times there is a lesion in continuity (axonotmesis) with spontaneous recovery only after six or twelve months.

External popliteal paralysis—A similar traction nerve injury may complicate severe varus strains of the knee when the structures on the outer side of the joint are torn or stretched. The external lateral ligament is avulsed with or without the styloid process of the fibula and severe traction of the external popliteal nerve may cause paralysis.

Circumflex and other palsies in shoulder dislocations—In a series of 571 shoulder injuries there were 231 dislocations of the joint with 34 nerve lesions and 340 fractures of the upper end of the humerus with no nerve lesions.¹ The nerve injury is obviously due to traction and the gross displacement of a dislocation is the most dangerous bone injury. The head of the humerus is usually dislocated forwards. The circumflex nerve and the posterior cord of the plexus winding from the back of the axilla to the outer side of the arm are therefore most commonly involved. Less commonly there is paralysis of the ulnar nerve of the musculo spiral nerve or of the other cords of the plexus. The paralysis usually recovers spontaneously.

The trunks of the brachial plexus or the nerve roots themselves may be stretched or even avulsed by traction injuries in which the arm is pulled away from the trunk or the head is forcibly flexed to one side. When this injury is sustained during delivery of a child and one of the two common birth palsies results (Erb Duchenne palsy involving the outer trunk and Klumpke palsy involving the inner trunk) the rule that traction nerve lesions usually recover spontaneously still holds true. On the other hand the more violent injuries of adult life may actually avulse the nerve roots from the cord and the prognosis is then hopeless.

Laceration of nerve—*Median nerve*—Median nerve paralysis in supra condylar fractures is usually due to simple nerve contusion but occasionally the injury is more severe and the nerve is severed over the sharp lower margin of the main fragment of the humerus. Spontaneous regeneration is then more difficult for there is actual loss of continuity of the nerve trunk. The surgeon's suspicion may be aroused by the degree of displacement of the fracture the sharpness of the proximal fragment the completeness of paralysis and the delay in recovery. In such a case he should not wait for more than about three months. If there is still no sign of recovery the nerve should be explored and sutured.²

Musculospiral nerve—The musculospiral nerve in the middle third of the humerus is also vulnerable. The nerve lies in a groove without muscular protection and in actual contact with bone. All musculospiral nerve lesions in fractures of the humeral shaft must be regarded with suspicion. Although the usual injury is a lesion in continuity which recovers spontaneously within three or four months division of the nerve must be suspected when there is displacement of sharp fragments with complete paralysis which does not recover quickly.

2 Fractures with Secondary Nerve Injuries

Friction nerve lesions—Passive joint stretching may cause friction neuritis of the ulnar nerve with paralysis due to *xonotmesis*. A fracture or dislocation causes roughening of the floor of the post condylar groove, no

¹ Watson Jones. Fractures in the Region of the Shoulder Joint. *Proc. Roy. Soc. Med.* 1926 xxix 1061.

² Platt and Britton. *Recent Results of Operations for Injuries of Peripheral Nerves (mainly Gunshot Injuries)*. *Brit. Jour. Surg.* 1933 xi 333.

complication arises until forcible passive movements are practised, friction of the nerve on the roughened bone day after day gradually sets up a secondary traumatic neuritis. In other cases the nerve is bound down by periarticular adhesions, and its normal mobility is restricted. Even a single forcible flexion movement under anaesthesia may then damage the nerve and cause paralysis.

Late compression nerve lesions—It is doubtful whether compression of a nerve by the callus of a uniting fracture ever causes paralysis. Such cases have been described in the past, but the lesion was probably due to friction of the nerve over callus as the result of passive stretching. Late compression of a nerve by scar tissue may sometimes hinder recovery from a primary nerve lesion, it is on this assumption that the operation of neurolysis has been recommended. Proof is still lacking, however, that this operation has in fact, been responsible for the recovery of nerve lesions which would not otherwise have recovered.

Late compression by splints, plaster, crutches, tourniquets—*The external popliteal nerve* lies in a vulnerable situation where it winds round the neck of the fibula. A tourniquet should never be applied at this level because it is so liable to cause paralysis. The nerve may be compressed by splints, plaster or strapping, even the pressure of a simple bandage or viscopaste dressing may cause paralysis. Special care must be taken when applying an unpadded plaster cast to protect the nerve by means of a pad of felt. Moreover, the upper margin of a below knee plaster cast should be cut away to a level below the neck of the fibula in order to prevent friction and compression of the nerve during movement of the knee. *The musculospiral nerve* is injured in a similar way if a forearm plaster extends no higher than 3 or 4 in above the elbow. When the limb hangs by the side, the upper margin of the cast digs into the back of the arm and compresses the nerve against the humerus. If compression and friction continue, paralysis supervenes within a few weeks. *There is much greater safety if the plaster is carried to a higher level, just below the axilla.* **Crutch paralysis**—Inadequate padding of axillary crutches may cause musculospiral paralysis by compression of the nerve in the lower part of the axilla. Padding with sorbo rubber, wool or even an old shirt wound on the crutch and secured with bandage is sufficient to prevent this complication. **Tourniquet paralysis**—Any form of rubber or inelastic tourniquet is dangerous in the upper limb. Apart from the risk of gangrene (p. 129), there is a very high incidence of musculospiral paralysis, and sometimes of median and ulnar paralysis. The flat rubber Esmarch tourniquet is little safer than other types. Only a pneumatic tourniquet with pressure controlled at 200 mm. should be used.

3 Fractures with Delayed Nerve Injuries

Late ulnar palsy—Delayed neuritis arising many years after injury occurs only in one situation. A child sustains a fracture of the external condyle of the humerus. If treatment is imperfect the fracture fails to unite and an increasing cubitus valgus deformity develops (Figs 257-258). There may ultimately be 40° or 50° of deformity. The ulnar nerve is stretched round the inner side of the joint. The deformity develops so gradually, and there is normally such free mobility of the nerve, that paralysis seldom occurs.

until adult life when it is precipitated by strenuous exercise. Since the nerve lies behind the elbow it is stretched by flexion movement. But it is already stretched to its normal limit by the valgus deformity. The further stretching sustained during hard work causes paralysis ten or twenty years after the original bone injury. The symptoms are completely relieved if the nerve is transposed to the front of the joint so that tension is relieved.



FIG 257



FIG 258

Cubitus valgus following non union of external condyle fracture in childhood. Stretching of the ulnar nerve round the inner side of the joint causes delayed neuritis and paralysis about ten years after the bone injury.

Recurrent dislocation of the ulnar nerve—An injury to the internal epicondyle of the humerus may tear or stretch the fibrous roof of the post condylar groove or it may distort the bone so that the groove is unduly shallow. The ulnar nerve then slips forwards over the epicondyle with each flexion movement of the joint and jerks back into its groove when the joint is extended. A frictional neuritis due to the recurrent dislocation may cause ulnar paralysis developing several years after the initial injury.

MEDIAN PARALYSIS

Etiology—A primary neuritis may be due to contusion of the nerve in dislocations of the carpal lunate bone and in supracondylar fractures of the humerus and sometimes a traction injury is sustained in dislocations of the shoulder. The nerve is occasionally severed at the elbow by supracondylar fractures.¹ Secondary neuritis may be due to scar tissue compression, especially in cases of Volkmann's contracture and may follow passive stretching of the elbow.

Clinical features—There is loss of sensation in the three and a half finger area (Fig 259). Patients frequently sustain cigarette burns of the

¹ Platt: Peripheral Nerve Complications of Fractures. *Jour. Bone and Joint Surg.*, 1938, 403.

anæsthetic fingers and the loss of sensation of the index finger and thumb constitutes a serious disability. The thenar eminence is flattened owing to wasting and paralysis of the abductor and opponens pollicis and the thumb tends to fall to the side of the hand (ape thumb deformity—see Fig 241). When the nerve is injured at the elbow there is also paralysis of the pronator

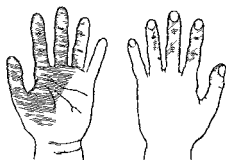


FIG 59

Area of anæsthesia in complete median paralysis

teres flexor carpi radialis flexor pollicis longus and part of the flexor digitorum profundus. But for the fact that flexion of the terminal joint of the thumb is impossible the functional loss due to paralysis of these muscles can be masked.

Treatment—Spontaneous recovery nearly always occurs within a few weeks in injuries of the wrist and within a few months in injuries of the elbow and shoulder. It is usually unnecessary to explore the nerve and the only treatment needed is electrotherapy of the affected muscles with a small splint to hold the

thumb in opposition and prevent stretching of the thenar muscles and an ape thumb deformity. Caution is necessary however in supracondylar fractures of the humerus particularly when the small distal fragment is displaced far backwards. Complete division of the median nerve sometimes accompanies rupture of the brachial artery in supracondylar fractures (p 123).

ULNAR PARALYSIS

Etiology—Ulnar paralysis seldom complicates injuries of the shoulder or wrist but traction lesions are common in injuries of the elbow. It may occur in avulsion of the internal epicondyle subluxation of the elbow with inclusion of the epicondyle or a dislocation of the elbow supracondylar fracture with outward displacement supracondylar fracture with forward displacement and in forward dislocation with fracture of the olecranon (Figs 252-256). Complete division of the nerve is practically unknown but severe stretching is possible and it may even be caught between the articular surfaces of the elbow on its inner side together with the displaced epicondyle. Secondary neuritis may follow passive stretching or manipulation of the joint. Delayed neuritis occurs in external condyle fractures and in recurrent dislocation of the nerve.

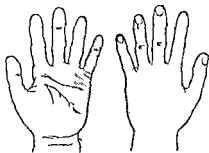


FIG 60

Area of anæsthesia in complete ulnar paralysis

Clinical features—In a complete lesion there is anæsthesia of the one and a half finger area (Fig 60) and paralysis of the interossei muscles the flexor brevis pollicis and adductors of the thumb. Paralysis of the interossei which normally flex the metacarpophalangeal joints

and extend the interphalangeal joints causes the typical claw hand (Fig 241)

Very often the lesion is incomplete. In mild cases of traumatic neuritis there is no more than tingling on the inner side of the palm. There may be a localised patch of anaesthesia or slight wasting or weakness of the intrinsic muscles of the hand (Fig 261). The classical test for muscle paresis is to estimate the power of gripping a sheet of paper between the sides of the fingers. This power is difficult to assess and the test is unreliable. It is better to ask the patient to grip a piece of wood or cardboard with the two thumbs and try to hold them flat (Fig 262). The least trace of weakness of the adductor pollicis and short muscles inserted into the proximal phalanx is at once obvious: they are overpowered by the long flexor inserted into the terminal phalanx, and the patient cannot prevent the interphalangeal joint from flexing (Froment's sign).

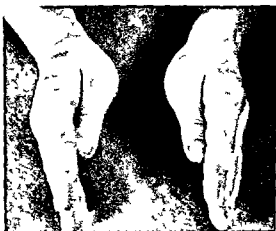


FIG 261

Incomplete ulnar paralysis. Note the slight wasting of the first dorsal interosseous muscle of the left hand.

Treatment—Expectant treatment is usually successful and recovery may be expected within six or twelve months. Electrical and massage treatment and active exercises are practised. Clinging and contracture of the fingers should be prevented by gentle stretching carried out by the patient himself. Passive stretching, forcible movements and manipulation of the elbow must be avoided. If after epicondylar and supracondylar fractures there is irregularity and distortion of the post condylar groove, anterior transposition of the nerve may be advisable.¹ This is also necessary in delayed ulnar palsy due to cubitus valgus and in recurrent dislocation of the nerve.



FIG 262

Incomplete ulnar paralysis. The patient cannot keep his thumb flat, owing to weakness of the short thumb muscles and unbalanced action of the long flexor (Froment's sign). This is the most reliable test for ulnar paresis.

elbow joint is divided but muscular branches are preserved. The deep fascia and the superficial fibres of the muscles over the front of the inner

Anterior transposition of the ulnar nerve—It is most convenient to lay the patient face down with the shoulder abducted and internally rotated and the elbow flexed to the right angle. The nerve is dissected out in the lower 2 in. of the arm and the upper 2 in. of the forearm. The branch to the

¹ Platt. The Operative Treatment of Traumatic Ulnar Neuritis at the Elbow. *Scg Cyn Obs*, 1925, xlvii, 822.

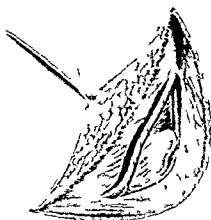


FIG 263

Anterior transposition of the ulnar nerve

condyle of the humerus are divided sufficiently to bury the nerve. The internal intermuscular septum is divided where it is crossed by the nerve in its new track (Fig 263). The aponeurosis of the flexor muscles is then lightly sutured over the nerve and the wound is closed.¹

MUSCULO-SPIRAL PARALYSIS

Etiology—The posterior interosseous nerve usually escapes in fractures of the neck and head of the radius but the musculo spiral trunk may be injured in supracondylar fractures, fractures of the shaft of the humerus and in dislocations of the shoulder. The pressure of inadequately padded crutches may cause transitory paralysis.

Clinical features—There is complete wrist drop due to paralysis of the extensors of the wrist, thumb and metacarpophalangeal joints of the fingers. The interphalangeal joints can still be extended by the interosseous and lumbrical muscles. The triceps muscle escapes for the branches which supply this muscle arise in the axilla. The sensory loss is unimportant and is confined to a small area on the radial side of the dorsum of the hand.

Treatment—The prognosis is very good indeed. Simple lesions due to contusion always recover, and even when the nerve is completely severed and is sutured permanent paralysis is exceptional. In some cases of badly infected compound fracture of the humerus where nerve suture has had to be delayed for six or twelve months and where at operation the nerve trunk was found reduced to a miserable strand suture has been rewarded by complete recovery (Fig 264). An attempt should always be made

however difficult the operation may be. Stretching of the paralysed muscles should be prevented by supporting the wrist in moderate dorsiflexion on a



FIG. 264

Infected compound fracture shaft of humerus with musculo spiral palsy. The severed nerve was sutured nine months after injury. Recovery is almost perfect.

short cock up splint or plaster cast. A special splint may be used with elastic strips on the backs of the fingers to assist extension movement. ¹ but a full length cock up splint or plaster which immobilises the fingers in extension should never be used. Such a splint often causes permanent stiffness of the fingers and this is a much worse disability than permanent paralysis. paralysis can be treated successfully by muscle transplantation where as no treatment will cure the crippling of permanent finger stiffness.

For the very few cases of irreparable nerve injury in which paralysis is permanent an excellent tendon transplantation is available. The pronator radii teres is stitched to the adjacent radial extensor tendons of the wrist the flexor carpi ulnaris is passed subcutaneously round the ulna and stitched to the extensor tendons of the fingers and the flexor carpi radialis is passed round the radius and stitched to the three extensor tendons of the thumb. Very satisfactory function is restored (Figs 265, 266).



FIGS. 265, 266

Result of tendon transplantation for irrecoverable musculo spiral paralysis in compound fracture of the humerus. The pronator teres flexor carpi radialis and flexor carpi ulnaris are transplanted to the extensors of the wrist thumb and fingers.

CIRCUMFLEX PARALYSIS

Etiology—The circumflex is the nerve most commonly injured in dislocations of the shoulder. The proximity of the nerve to the neck of the humerus its limited mobility and its course from the back of the axilla to the outer side of the humerus explain its vulnerability. Rather more than 5 per cent of shoulder dislocations are complicated by circumflex palsy.

Clinical features—The area of sensory loss is unimportant but there is complete paralysis of the deltoid. This can be tested on the first day of injury without actually moving the shoulder by palpating the muscle with the fingers of one hand and asking the patient to attempt gentle abduction against the resistance of the other hand which is placed over the elbow. Circumflex nerve lesions are to be distinguished from injuries to the posterior cord of the plexus (where there are the clinical features of combined circumflex and musculospiral paralysis) and from injuries to the outer trunk of the plexus (paralysis of deltoid and of biceps).

Treatment—Stretching of the deltoid must be prevented by supporting the limb in an abduction frame. If this is applied within a few days of injury, great care must be taken to prevent redislocation by bandaging the frame securely to the shoulder and trunk. Some surgeons defer the application of the frame for three or four weeks. The splint is worn day and night and the arm is never lowered below the right angle until recovery is sufficient to enable the patient to raise the limb actively off the frame.

Recovery is usually complete in from two to six months. During a five year period I observed 15 cases of circumflex palsy in dislocations of the shoulder, of which 10 recovered within six months, 3 within twelve months and 2 were permanently paralysed.¹

SCIATIC PARALYSIS

The sciatic nerve is sometimes injured in posterior dislocations of the hip and rarely in fractures of the pelvis. Traction injuries may be sustained during attempts at manipulative reduction of hip dislocations. The lesion is usually incomplete and the external popliteal fibres are chiefly involved. In a complete lesion there is paralysis of all muscles below the knee and anaesthesia below the knee except in the area supplied by the long saphenous nerve on the inner side of the leg. Trophic lesions and traumatic ulceration of the foot may occur. The prognosis after complete sciatic palsy is grave but incomplete lesions due to contusion usually recover.

LATERAL POPLITEAL PARALYSIS

Etiology—The nerve may be stretched in rupture of the external lateral ligament, avulsion of the styloid process of the fibula and dislocation of the knee.^{2,3} Secondary lesions arise from compression of the nerve against the neck of the fibula.

Clinical features—There is paralysis of the anterior tibial and peroneal

¹ Watson Jones, *J. Roy. Soc. Med.* 1936, xxix, Section of Orthopaedics, 24.

² Hault, *Ext. Popliteal Nerve Inj. cont. in Fract. of Fibula*, *Jour. Roy. Soc. Med.* 1928, x, 419.

³ Watson Jones, *Ext. Popliteal Nerve Inj. cont. in Fract. of Fibula in Knee Joint with Lateral Dislocation*, *Jour. Bone and Joint Surg.* 1931, xi, 2, 28.

muscles and anæsthesia of the outer aspect of the leg and dorsum of the foot

Treatment—Mild contusion and compression injuries recover spontaneously within a few months. Stretching of the paralysed muscles must be prevented by a plaster cast with *the foot fully dorsiflexed and in slight eversion*. After weight bearing is resumed an iron with a drop foot stop should be worn until recovery is complete. The rare traction injuries are more serious. Early exploration is advisable since recovery after delayed suture is exceptional.

CHAPTER VIII

CLINICAL AND RADIOGRAPHIC DIAGNOSIS OF FRACTURES

CLINICAL DIAGNOSIS

In many bone and joint injuries the clinical features are obvious. Simple inspection reveals the classical signs of local swelling, ecchymosis, deformity and inability to use the limb. No attempt must be made to elicit the other classical sign—crepitus due to grating of the fragments on each other. Manipulation for the purpose of diagnosis is unnecessary. It causes unnecessary pain and it may be responsible for damage to the blood vessels or nerves. Even when there are no obvious signs, an adequate diagnosis can be usually made on inspection and palpation alone. *There is no necessity to move the limb at all.* The position and contour must be compared with the normal limb. When an elderly patient falls and injures the hip, slight external rotation deformity is sufficient to make a provisional diagnosis of fracture of the femoral neck even in the absence of other clinical signs. Flattening of the contour of the shoulder may disclose a dislocation of the joint. Slight prominence of the lower end of the ulna shows a fracture of the radius and so on.

Localised bone tenderness—The one sign which requires emphasis, because it is sometimes ignored, is persistent local tenderness over one part of the bone. In many impacted fractures, crack fractures and greenstick fractures, this may be the only clinical sign. If there is localised bone tenderness a fracture must be assumed until it is disproved by radiographic examination. Fractures of the carpal scaphoid bone are often overlooked, and prolonged or even permanent disability caused, by attributing tenderness over the radial side of the wrist to a simple sprain. *Reputations have been ruined by attempting to differentiate with certainty between a sprain and a fracture of the ankle without the aid of radiography.*

Diagnosis of injury to soft parts—When it is established that there is a fracture, an equally important part of the clinical examination still remains. The limb must be examined for injury to the vessels and nerves. In fractures of the spine a careful neurological examination is essential, and in thoracic and pelvic injuries visceral damage must be excluded.

RADIOGRAPHIC DIAGNOSIS

No attempt should be made to reduce the displacement until good X ray films are available. Contrary to a curious belief, this rule applies just as forcibly to dislocations as to fractures. I have seen not one but

The patient whose radiographs are shown below in Fig 268 has sustained a backward dislocation of the ankle with a fracture of the posterior articular margin of the tibia. The lower end of the fibula is displaced back with the astragalus. There must almost certainly be a fracture of the fibula at the junction of the displaced lower shaft with the undisplaced upper shaft.

Although half of the fibula is included in these radiographs there is no evidence of fracture. Further radiographs must be taken to include the whole length of the fibula.

Raise flap to see Fig 267

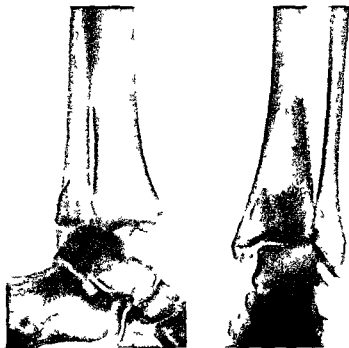


Fig 268

Radiographs of the ankle joint (Fig 268) show a backward dislocation and a posterior marginal fracture of the tibia but no fracture of the fibula. This displacement could not arise without a fracture of the fibula. The upper half of the fibula must be X-rayed (Fig 267).

many fractures of the neck of the humerus which have been twisted in all directions under the impression that the injury was a dislocation. Even the most expert surgeon may be unaware that an elbow dislocation is complicated by avulsion of the internal epicondyle, and that there is danger of inclusion of the fragment within the joint. Radiography does more than confirm the diagnosis and warn the surgeon of difficulties, it indicates the exact type of manipulation and the degree and direction of force which is necessary to reduce the displacement. The film should be in full view throughout the manipulation.

Elements of radiographic diagnosis—Screening is unreliable and films of the best quality must always be taken. No diagnosis should be attempted



FIG 269

A surgeon accepted this film and believed that the injury was a simple fracture of the ulna needing no treatment other than the application of plaster

Is this a simple fracture of the ulna (Fig 269)? Is there any other injury to the forearm? The situation of the fracture should itself make the surgeon suspect the probability of an associated dislocation of the head of the radius. But diagnosis must be based on evidence not suspicion. The nearest joint should have been included in the film.

Raise flap to see Fig 270

unless there is good contrast between bone and soft tissues, unless the injured region is in the middle of the film so that the whole of the suspected area is covered and unless at least two views in planes at right angles to each other are available. It is better to have no X ray at all than to rely on one film (Figs 271-274). A fracture may be obliquely across the bone so that the radiographic shadows of the fragments overlap and mask the injury and the displacement which is quite evident in other planes. Radiographs of fractures of long bones must always include the joint above or below the injury (Figs 269-270). A short film which covers only the shaft is usually worthless because it may be impossible to know the direction of displacement. Furthermore in injuries of the forearm and of the leg, a fracture of the shaft



FIG 271

The injury appears trivial. The slight splintering of the malleoli looks to be unimportant. It would appear that no special treatment is needed.

A patient has sustained an injury to the ankle joint. The anteroposterior radiograph seen in Fig 271 shows very slight splintering of the malleoli but no evidence of complete fracture, dislocation or of any displacement. Can this evidence be accepted? Is the injury as trivial as it appears to be?

Raise flap to see Fig 272



FIG 273

The anteroposterior radiograph suggests that if there has been any fracture of the neck of the femur at all, it is impacted in perfect position. The surgeon responsible for this case did not believe from this evidence that any special treatment was needed.

An elderly patient stumbled and complained of pain in the hip. There was no definite physical sign of injury but the patient lay with the limb in external rotation. The routine anteroposterior radiograph is seen in Fig 273. Can it be accepted that there is no serious injury to the hip?

Raise flap to see Fig 274

of one bone may be accompanied by an injury to the other bone at an entirely different level. If there is a fracture of the shaft of the tibia with overriding and no evidence in the film of injury to the fibula the whole length of the fibula must be radiographed. There is probably a fracture of the neck of the bone. A backward dislocation of the ankle may be accompanied by a fracture of the upper third of the shaft of the fibula (Figs 267-268). A fracture of the shaft of the ulna is sometimes associated with a dislocation of the head of the radius (Figs 269-270) or a fracture of the shaft of the radius with a dislocation of the inferior radio ulnar joint.

SPECIAL RADIOGRAPHIC EXAMINATIONS

Three types of special radiographic examination are important in diagnosis: (a) radiographs in three planes or in four planes; (b) repeated radiographic examination after an interval; (c) radiographs in different positions of the joint and in the position of deformity.

Radiographs in three planes—It has been generally assumed that if radiographs are taken in two planes at right angles to each other no bone injury



FIGS 273-276

Anteroposterior and lateral radiographs show no evidence of a fracture of the carpal scaphoid bone but these views do not exclude such an injury.

FIG 277

A three-quarter oblique view clearly shows a fracture of the tubercle of the scaphoid.

or displacement can escape notice. This is erroneous. A fracture may lie in a plane of such obliquity that the shadows of the fragments overlap accurately in both the classical positions. The injury is disclosed only when a film is taken in the oblique axis. This very often applies to fractures of the carpal scaphoid bone (Figs 273-276). Fractures of the wrist of the scaphoid and of the tubercle of the scaphoid may be masked in strict anteroposterior and lateral views even if the radiographs are examined stereoscopically. Furthermore a fracture of the waist of the scaphoid which has been immobilised may appear to be united when examined only in anteroposterior and lateral views and yet an oblique view shows that union is unsound. It should be a routine practice in radiography of the scaphoid bone to take at least three views.

Marginal chip fractures of the back of the lower end of the radius may involve the groove for the extensor pollicis longus tendon. If the wrist is not immobilised the tendon gradually wears and frays over the sharp edges of the fractured bone until it undergoes complete spontaneous rupture,

if it is immobilised in plaster the complication is usually avoided. Radiographic diagnosis is therefore most important and yet the injury is seldom seen in either antero posterior or lateral views. If the injury is suspected by the clinical sign of tenderness on pressure over the bone and routine radiographs fail to show it other views should be taken in the two oblique planes (Chapter XXVI).

Fractures of the lateral malleolus may not be visible in the antero posterior radiograph and in the lateral plane the shadow is overlapped by that of the tibia. Radiographic evidence of early union cannot be secured by either of these views. A third film should be taken in the oblique axis.

Repeated radiographic examination after an interval—If a radiograph fails to show evidence of a fracture which is suspected strongly on clinical grounds the examination should be repeated after two or three weeks. If there is a fracture movement of the fragments during the interval causes traumatic hyperæmic decalcification so that the inconspicuous crack becomes an obvious fracture. This is particularly important in suspected fractures of the carpal scaphoid bone where the crack may be so fine that



FIG 278

There is no definite radiographic evidence of any fracture of the scaphoid bone

A patient sustained a wrist injury and the clinical features suggested a fracture of the scaphoid. Fig 278 is the antero posterior radiograph. Lateral and oblique views showed no abnormality. Is there a fracture of the scaphoid? Can a fracture be excluded on this radiographic evidence?

Read flap to see Fig 279

even good films do not demonstrate it (Figs 278-279 and Chapter XXVI). Similarly in suspected march fractures of the metatarsal bones radiographs taken within a day or two of the onset of symptoms often fail to show the fracture whereas after two or three weeks the line of fracture is obvious and there is also subperiosteal callus formation. The same procedure of repeated examination after an interval is often of value when the surgeon is not certain whether union of a fracture is sufficiently firm to discontinue immobilisation with safety (see p. 161).

Slight crush fractures of a vertebral body may undergo spontaneous reduction by alteration in the posture of the patient. The injury is produced by flexion of the spine. If the spine is then fully extended the displacement is sometimes reduced so perfectly that no radiographic evidence remains. Unless the spine is immobilised in extension the damaged vertebra will slowly collapse and be compressed by weight bearing strain. A second radiographic examination ten to fourteen days after the first will show the

first stages of this recurrent wedging and confirm the diagnosis which could not be established by the original examination

Radiographs in different positions of the joint—Certain bone and joint injuries are concealed in routine radiographs and disclosed only when radiographs are taken with the joint in a position of strain

Sprains and subluxations of the ankle joint—For many years it was believed that dislocation of the ankle joint unaccompanied by fracture was exceedingly rare. It was also believed that sprain of the ankle joint was a minor injury which could be treated safely by strapping support and early mobilisation this view being held despite the conviction of many patients that a sprain was more serious than a fracture. Actually dislocation of the ankle joint occurs frequently but the displacement is momentary and it undergoes spontaneous reduction the clinical signs then suggest a simple sprain and radiographs show no evidence of bone or joint injury (Fig 280). Only radiographs taken with the foot held in the fully inverted position show tilting of the astragalus within the tibio fibular mortice thus proving momentary dislocation of the joint and complete rupture of the lateral ligament (Fig 281). This injury necessitates immobilisation in plaster for not less than eight or ten weeks. If it is treated as a simple sprain by early mobilisation the ligament fails to unite the joint remains unstable recurrent dislocation develops and the astragalus subluxates every time the foot is inverted. The patient may fall to the ground he dare not walk freely over rough irregular surfaces and the foot constantly gives way. In former years when routine radiographic examination was relied upon the diagnosis was still obscure and it is not surprising that patients who travelled from one doctor to another and from doctors to osteopaths and bone-setters who were treated by massage electrotherapy and manipulation and who still gained no relief from their disabilities believed that sprain of the ankle was a most serious injury.

The diagnosis of momentary subluxation and the sequel of recurrent dislocation of the ankle joint was described in the first edition of this book and since that time the technique of radiographic examination of the fully inverted foot has been accepted. The frequency of the injury is now recognised. Rowland Hughes¹ points out that in 50 per cent of sprained ankles slight tilting of the astragalus can be demonstrated and in 20 per cent of injuries there is considerable tilt which amounts to subluxation of the joint. A clear distinction must therefore be drawn between sprains due to stretching of fibres of the anterior fasciculus of the lateral ligament which may be treated safely by novocaine injection massage or simple elastic support and momentary dislocations with rupture of the middle fasciculus and tilting of the astragalus in which complete immobilisation in plaster is necessary. The radiographic test is not reliable unless local novocaine anaesthesia is used because otherwise the patient resists the painful movement by strong peroneal spasm. Moreover the surgeon should hold the foot himself and not rely on radiographers who are afraid of causing further injury and may fail to demonstrate the displacement because they do not invert the foot strongly enough. Finally whatever grip the surgeon may choose to employ he must invert the heel and not simply adduct the forefoot.

Rupture of the medial ligament of the ankle joint—In the same way that



FIG 280

Severe sprain of the ankle There is no radiographic evidence of bone or joint injury

The radiographs shown above (Fig 280) are of a patient who sustained a severe inversion injury of the ankle joint. There was swelling, ecchymosis, and tenderness on pressure over the external lateral ligament.

Routine radiographs show no fracture or dislocation. Is the injury therefore a simple sprain of the ankle which will recover fully after strapping the joint for two or three weeks?

Raise flap to see Fig 281

rupture of the lateral ligament is concealed unless radiographs are taken of the inverted foot rupture of the medial ligament may be concealed unless radiographs are taken of the everted foot This routine is important in undisplaced subperiosteal fractures of the lateral malleolus if it is proposed to adopt the treatment of novocaine injection and immediate unprotected weight bearing Such treatment may be safe if the fracture is the isolated injury it appears to be but routine radiographs show no distinction between isolated fractures of the lateral malleolus and fractures of the malleolus with rupture of the medial ligament and potential outward dislocation of



FIG 282

The classical antero posterior and lateral views of the cervical spine show no evidence of bone or joint injury

After a flexion strain of the neck the patient whose radiograph is shown in Fig 282 complained of pain radiating down both arms and of tingling in the fingers There is arthritis at the fifth cervical level shown in the lifting of the margins of the vertebral body

Is there any evidence of injury? Can a dislocation or subluxation of the cervical spine be excluded?

Raise flap to see Fig 283

the foot Before unprotected weight bearing is permitted radiographs must be taken with the foot held in full eversion preferably after novocaine injection of both malleolus and ligament

Inferior tibio fibular diastasis—Even when there is no fracture it is possible for the astragalus to subluxate outwards if rupture of the medial ligament of the ankle joint is accompanied by rupture of the inferior tibio fibular ligament This subluxation is no less important a source of permanent disability than Pott Dupuytren fracture dislocations of the joint the displacement must be reduced and the joint must be properly immobilized Every severely sprained ankle should therefore be X rayed with the foot held both in full inversion and full eversion

Rupture of the inferior tibio fibular ligament with diastasis of the joint may also occur as a complication of abduction fracture dislocations of the

ankle joint It is difficult to prevent permanent disability because redisplacement often occurs despite the protection of a carefully moulded plaster cast, the added fixation of a screw which fixes the tibio fibular joint is often necessary. The diastasis may be concealed in routine radiographs because the joint is obscured by the overlapping shadow of the tibia. It is therefore advisable to take radiographs with the foot held in maximum eversion, if the gap on the inner side of the joint between astragalus and medial malleolus is greater than can be accounted for by displacement of the lateral malleolus diastasis of the inferior tibio fibular joint is proved and steps must be taken accordingly (Chapter XXXII)

Subluxation of the cervical spine—A suspected fracture or dislocation of the spine which is not shown in routine radiographs may be disclosed by repeating the lateral examination with the spine in a moderate degree of flexion. This routine was of great importance to the patient whose radiographs are shown in Figs 282 283. He was sitting in a vehicle which stopped suddenly so that his head was jerked sharply forwards. There was no direct injury but he complained of severe pain in the neck and down both arms. Radiographic examination showed no bone or joint injury and the history of accident was vague. After six weeks he was accused of malingering his compensation was stopped and he was told to resume heavy work. The distribution of pain suggested a possible subluxation of the cervical spine with root pressure and it proved easy to confirm this by repeating the radiographic examination with the spine in flexion (Fig 283). There is obviously an incomplete dislocation of the interarticular and intervertebral joints which appears only in the flexed position. Immobilisation in plaster was necessary for ten weeks to allow tightening of the damaged ligaments, and the man ultimately returned to full work despite the arthritic change.

DIAGNOSIS OF UNION OF A FRACTURE

Clinical evidence of union—The diagnosis of union of a fracture should be based on clinical tests—freedom from elasticity and springing absence of pain when angular strains are applied and disappearance of tenderness on deep pressure. These clinical signs may show evidence of union long before there is radiographic evidence of calcification of the callus (for example in fractures near the lower end of the radius supracondylar fractures of the humerus fractures of the clavicle and fractures of the ribs). The radiologist must bear this in mind when reporting on radiographs taken many weeks after injury particularly when his opinion may be of medico legal importance. He should not report that the fracture is ununited unless there is sclerosis of the fractured surfaces proving established non union. In earlier cases his opinion is better expressed in the words 'there is not yet radiographic evidence of consolidation'.

Importance of radiographic evidence—In certain fractures it is important that clinical tests of union should be supplemented by radiographic evidence before splints or plaster are discarded. In fractures of the carpal scaphoid bone no clinical test of union is reliable and the surgeon must rely entirely on X ray evidence. In fractures of the shafts of the leg bones clinical examination may appear to show firm union at a time when unprotected

weight bearing would be followed by yielding of the fracture. This was a frequent cause of non union and mal union in former years when patients were discharged from hospital as soon as union appeared firm to clinical tests and often within six or eight weeks of injury. In such fractures the diagnosis of union should be based on the combined evidence of clinical and radiographic signs.



Fig 284

The fracture appears to be firmly consolidated and it would appear that union is so sound that any degree of weight bearing strain could be sustained with safety.

A fracture of the shafts of both leg bones has been immobilised in plaster for several months. Antero posterior and lateral radiographs appear to show sound consolidation of the fracture (Fig 284). The film is under exposed. There is no clear differentiation of cortex from medulla. Bone striæ are not visible. Is this evidence sufficient?

Raise flap to see Fig 285

Radiographic evidence of union—Union of a fracture may be accepted when there is a continuous external bridge of callus joining the fragments or when callus between the fragments is uniformly calcified and of a density approaching that of normal bone. This evidence must be interpreted with care. No film should be accepted unless it is correctly exposed, properly developed and with no light fog so that there is minimal soft tissue shadow, a bright bone shadow and clearly visible bone texture. An underexposed film with a heavy soft tissue shadow may give an appearance of calcification of callus which is actually due to overlying soft tissues (Figs 284-285). The possibility of overlap of bone shadows must also

be excluded. A single radiograph is often misleading (Figs 286 287). Even two views at right angles may cause error if neither view coincides with the plane of an obliquely placed fracture. This is nearly always the case in fractures of the carpal scaphoid bone and one or more oblique views must be taken routinely. In other fractures it is often wise to supplement the traditional antero posterior and lateral radiographs with oblique views.

If there is doubt the radiographic examination should be repeated with angulatory strains applied to the fracture site. In a fractured tibia for



FIG 286

In the antero poster or radiograph union appears sound

A patient sustained a basal fracture of the neck of the femur. These fractures almost invariably unite soundly by bone and non union is very exceptional. Six months after injury antero posterior radiographs appear to show good union (Fig 286). Can we be sure from a single radiograph that union is in fact consolidated? Is it possible that the appearance of union could be simulated by overlap of bone shadows?

Raise flap to see Fig 287

example a film is taken while the limb is held with varus strain and compared with one taken with valgus strain. Difference in the alignment of the fragments in the two films showing elastic springing of the fragments proves that union is not sufficiently firm for unprotected weight bearing. It is often advisable to draw a line on the film in the axis of each fragment so that alignment can be judged more accurately. Figs 288 290 show a slightly different application of this test. A fracture of the shaft of the femur had been treated elsewhere by means of the Haines transfixion apparatus (see p 20). It was suspected that the apparatus was not only causing persistent distraction of the fragments but that it was also failing to immobilise them adequately so that angulatory movement was occurring despite the transfixion pins. The clinical suspicion was at once confirmed when radiographs were taken with firm pressure applied first to the outer side and then to the inner side of the thigh.

The most important check in cases where radiographic evidence is inconclusive is to insist on repeating the X ray examination after an interval of three or four weeks. This precaution is of particular importance in



FIG 288

Fracture shaft of femur treated by Haines transfixion apparatus. It was suspected that angulatory movement was occurring.

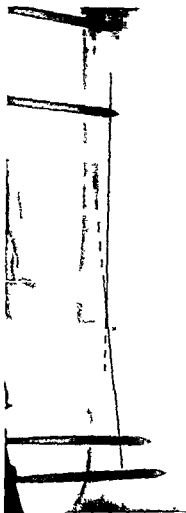


FIG 289

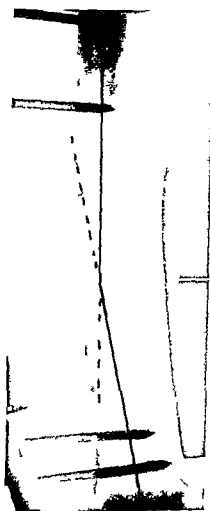


FIG 290

Radiographs taken with angulatory strain.

In Fig 289 pressure is being applied to the outer side of the thigh and in Fig 290 to the inner side. The altered alignment proves that the apparatus is not immobilizing the fragments completely. The fracture has been distracted and is together with inadequate immobilization is causing non union of the fracture.

fractures of the carpal scaphoid bone and in lower limb fractures where it has been decided to discard the plaster and allow weight bearing. After an interval of three or four weeks unsound union is shown by decalcification of the callus if the density of calcification has increased despite the lack of protection union is sound.

Radiographic evidence of consolidation—Final consolidation of a fracture is shown by (1) uniform and uninterrupted calcification of callus which is now even more dense than normal bone (2) absorption and consolidation of external callus and (3) development of continuous striæ across the fracture site. The diagnosis may be confused however exactly like the



FIG 291

Old fracture of external condyle of humerus. Is the union firm enough to prevent increasing valgus deformity or not?

The radiograph shown in Fig 291 is of a boy aged twelve who injured the joint six years ago and now has no symptoms. The parents ask for a prognosis. Will the elbow injury interfere with an Army career?

Is the external condyle fracture united by bone or is it merely a fibrous union which will permit an increasing valgus deformity and lead ultimately to delayed ulnar palsy?

Raise flap to see Fig 292

earlier diagnosis of union by overlapping bone shadows. I was recently asked for an opinion on a six months old fracture of the shaft of a fifth metacarpal near its base. Two surgeons said that it was united and two said that it was ununited. Radiographs appeared to show consolidation, but in all views there was overlap of shadows of the fragments. Clinical tests were unreliable because it was difficult to judge whether movement was occurring only at the carpo metacarpal joint or whether there was also movement at the site of fracture. Radiographs taken with forward and with backward strain showed considerable angulation of the fragments and proved non union of the fracture. Similarly Fig 291 shows an old fracture of the external condyle of the humerus in which it is difficult to be certain of the

degree of union Will this remain a stable elbow and continue to give good service free from symptoms and from complications or is it an unstable elbow which will develop an increasing cubitus valgus deformity and ultimately lead to delayed ulnar palsy? A radiograph taken with the elbow held with the greatest possible valgus deviation of the forearm gives the answer (Fig 292) The union is unsound

Difficulties in the diagnosis of final consolidation of a fracture may also arise when hair line reduction of a fracture has been secured by operation and maintained by plates or screws particularly for example in ruled fractures of the neck of the femur where there is little or no external callus In these fractures the greatest caution is needed before making a decision that union is finally consolidated and that nails or screws may be removed

THE DANGER OF X-RAYS TO SURGEONS

Many surgeons have wrecked their careers by fluoroscopic screening of fractures radiodermatitis of the hands has made it impossible to scrub up, chronic ulceration has needed surgical excision and plastic repair fingers have been amputated cancer has developed^{1 2} This is not an idle threat It is what actually happened to ninety one surgeons who used fluoroscopy for reducing fractures^{3 4} The figure relates only to published cases in one locality it does not include radiodermatitis from causes other than fracture treatment it does not include milder cases of erythema fissured nails rough skin slight telangiectasis pigmentation atrophy of the fat pads and hang nail it includes only severe cases of indurated board like skin acute and chronic ulceration and cancer

If a surgeon puts his hands in the direct beam of radiation the daily tolerance dose is exceeded in three seconds⁵ He cannot possibly reduce a fracture in three seconds and his only alternative is to use lead lined gloves a handicap with which it is almost impossible to manipulate fractures successfully I have never met a surgeon practising the manipulative reduction of fractures under fluoroscopic screens who will swear that he never removes his gloves at a critical stage of the manipulation If gloves are removed for no more than a few seconds and the surgeon treats no more than four or five fractures a week he is doomed He will learn his fate only when it is too late He will regret the day that he permitted himself to use a dangerous practice—dangerous not only to himself but also to his team

Even if the surgeon does not put his hands in the direct beam and uses no more than intermittent fluoroscopy by which to check the result of his manipulations the daily tolerance dose from scattered radiation is reached within ten minutes I would prohibit every surgeon from using the fluoroscope in reducing fractures Equally good control is available by the use of ordinary films developed rapidly and examined within two or three minutes Even with this procedure the surgeon may be exposed to scattered radiation Moreover he is also committed to the occasional risk of holding

¹ *Clinical Orthopaedics* 1941, 3, 101. ² *Brit. Med. Jour.* 1941, 11, 101.

³ *Brit. Med. Jour.* 1941, 11, 101.

⁴ *Brit. Med. Jour.* 1941, 11, 101.

⁵ *As the average sensitivity is 30 mR per 1% reduction in film density, giving a clearance of 1% in density, the tolerance dose of scattered radiation is reached in the direct beam in 3 sec. at 40 in. distance and in 30 in. at 10 in. distance. (The tolerance dose of scattered radiation is 30 mR per 1% reduction in film density.)*

a joint or fracture in a position of strain while radiographs are taken and although lead lined gloves must be worn almost invariably, this protection is sometimes impossible (as for example in testing for non union of a small bone like the fifth metacarpal or as in the case shown in Fig 289 where the glove was too thick to be introduced between the apparatus and thigh) Finally the surgeon is committed to the use of radiography during open



FIG 289

The result of exposure to X rays. Ninety one surgeons are known to have paid this price. (By courtesy of the London Hospital and Dr. Jope.)

operations when he cannot use lead gloves, this alone may be dangerous, and mechanical devices must be employed for holding the cassette during nailing operations on fractures of the neck of the femur and grafting operations on ununited fractures of the carpal scaphoid bone. The very rare occasions when protection is impossible must be the limit of risk which the surgeon will accept.¹ Fluoroscopy should not be used. The temptation is great. The risk is greater. The advantage is negligible. Ninety-one surgeons are known to have paid the price. Don't be the ninety-second!

¹ Use of X rays in the Manipulation of Fractured Limbs. Report of British X ray and Radium Protection Committee. *Brit Med Jour* Jan 17 1947 84

CHAPTER IX

REDUCTION AND IMMOBILISATION OF FRACTURES

Two of the three main principles of fracture treatment, the principles of immobilisation and of functional activity, were discussed in Chapters II and III. The third concerns reduction of the displacement. These principles are of equal importance and each of them must be observed throughout all stages of treatment—

- 1 *Reduction*—Displacement of the fragments must be corrected and redisplacement prevented under radiographic control
- 2 *Immobilisation*—The fragments must be immobilised completely, continuously and without interruption until union is firm
- 3 *Functional activity*—Joints which need not be immobilised must be actively exercised but never passively stretched

The aim of treatment is both anatomical and physiological. Anatomically, to restore the bone to its former length, alignment and shape, physiologically, to restore joints, muscles, vessels and nerves to their former function. Physiological recovery is accelerated by early functional activity which encourages repair of the bone, maintains a normal circulation, preserves the tone of muscles and the movement of joints, promotes mental recovery and prevents psychological complications. The ideal, therefore, is 'early use of the injured part without movement of the injured structure'. But this functional activity is safe only if redisplacement can be avoided. Padded splints are inadequate. Closely fitting plaster casts accurately moulded to the contour of the limb are usually essential.

Methods of reduction and immobilisation—The four methods of reducing and immobilising fractures are—

- 1 Manipulative reduction and plaster immobilisation
- 2 Manipulative reduction and continuous traction
- 3 Mechanical reduction and skeletal transfixion
- 4 Operative reduction and internal fixation

MANIPULATIVE REDUCTION AND IMMOBILISATION IN PLASTER

The technique consists simply in inspecting radiographs to see where the fragments lie, replacing them by direct pressure or by traction, and repeating the radiographic examination to ascertain whether complete reduction has been secured. If reduction is not complete, the routine of manipulation, fixation, and X-ray examination is repeated. Reduction must be achieved by guarded measured strength rather than by sudden jerking force. Nevertheless in fractures near the ends of long bones considerable strength may be required. It is difficult to over reduce &

Colles' fracture of the radius but easy to under reduce it. In many fractures of the ankle and in epiphyseal separations the fragments are so shaped that they lock when fully reduced and it is impossible by manipulation to over reduce these injuries however strong the pressure which is applied.

In over riding fractures of the shafts of long bones traction is necessary. It is a common mistake to apply perfunctory traction for only a few seconds. Most fractured surfaces are irregular and projecting spikes prevent the fragments from sliding into position. The limb must be slightly overlengthened before the serrations disengage and slow steady traction must be continued for five or perhaps even ten minutes.

Manipulation under anaesthesia—To attempt to reduce any fracture without an anaesthetic is unfair to the patient and unfair to the surgeon. Even the oldest and most fragile patient can stand a few minutes of gas anaesthesia with safety. If for constitutional reasons it is believed that general anaesthesia is inadvisable a local anaesthetic may be employed.

Local anaesthesia for fracture reduction—In this country where general anaesthesia has reached a high standard of efficiency and safety, local anaesthesia is seldom used routinely. The analgesia is not always perfect in impacted fractures where diffusion is difficult or in fractures more than two or three days old where the haematoma is absorbing. It is not suitable for children or for nervous adults. Moreover dilution of the fracture haematoma may delay repair. On the other hand local anaesthesia has the advantage of lasting for several hours so that manipulations may be repeated when necessary. A long hypodermic needle is introduced through an intradermic wheal over the site of fracture. When blood can be withdrawn confirming that the point of the needle is in the fracture haematoma 10-20 c.c. of 2 per cent novocaine are injected slowly. Analgesia should be complete within ten minutes. If the fracture is impacted the novocaine must be injected subperiosteally at three or four points.

Standard of reduction—How perfectly must the fracture be reduced? The standard of end result must be a limb clinically indistinguishable from normal. It must have normal function and it must have a normal appearance. The alignment must always be perfect and there must be no rotational displacement. Slight loss of apposition of the fragments may be of no significance in fractures of the shafts of long bones especially in children. On the other hand more severe lateral displacement causes bony thickening and impairs the cosmetic result especially in a subcutaneous bone such as the tibia. Even 1 or 2 mm. of lateral displacement in a Colles' fracture of the radius produces an ugly deformity. Moreover lateral displacement may impair the stability of the fragments. As a rule therefore an anatomical reduction is necessary but some latitude is permissible in the apposition of fragments particularly in shaft fractures. No angulation is permissible even in the fractures of children.

Time of reduction—The reduction of fractures and dislocations may be performed at any time during the first few days after injury. Dislocations should always be manipulated at once because severe pain does not begin to subside until the displacement is reduced. With fractures there is often less urgency. Pain is relieved if the fragments are immobilised by first aid splints even if the displacement is not perfectly reduced.

If the fracture is manipulated within an hour or two of injury, before there is reactionary swelling great care must be taken not to apply so tight a plaster that the circulation might be endangered when the limb does swell. A plaster slab is applied over half or two thirds of the circumference of the limb and held in place with a soft bandage which may be cut if the necessity arises. The encircling plaster is completed the next day. If, on the other hand from twelve to twenty four hours has elapsed and the limb is already very swollen even a closely applied plaster will become loose after a few days when the swelling subsides. A new cast must therefore be applied during the second or third weeks. In some fractures of the ankle and leg which have not been elevated or supported by firm elastic pressure the limb may swell almost to twice its normal size. In such cases the routine of delayed reduction is preferable.

Delayed reduction of fractures with severe swelling.—Without preliminary manipulation and without anaesthesia the fracture is immobilised by a plaster slab. The patient is put to bed with the limb elevated. Two or three days later when the swelling has subsided the fracture is manipulated and a complete unpadded plaster cast is applied. This routine is not applied to dislocations or to fractures where the displaced fragments may exert pressure on nerves or vessels. Delayed reduction of supracondylar fractures of the humerus is permissible only if there is no complication and if the circulation is undoubtedly normal. Moore of Philadelphia has been so impressed with the frequency of early redisplacement that he invariably uses delayed reduction. No attempt is made to reduce a closed fracture before the third or fourth day. He finds that manipulation is facilitated and the incidence of secondary displacements is reduced.

Radiographic control of reduction.—However satisfied the surgeon may be with his reduction of a fracture there can be no excuse for neglecting to secure radiographic confirmation. A post reduction X ray must always be taken. It is true that the surgeon may decide to ignore certain displacements because he knows that the functional and cosmetic result will still be perfect but unless radiographic examination has made him fully aware of the displacements which exist he is not in a position to know whether it is safe to ignore them. I have seen several fracture dislocations of the ankle, treated even by expert surgeons where imperfect reduction was recognised so late that arthrodesis of the joint was the only available treatment.

If a limb is swollen at the time that the first plaster is applied check radiographs must be taken ten days later before the plaster is changed. The fragments may have been redisplaced already. Furthermore the limb must again be X rayed after applying the new plaster to ascertain whether the fragments still lie in the correct position. Every time that a plaster is renewed this routine must be practised however unlikely it may appear that the fragments have moved.

Certain fractures are prone to redisplacement within the plaster even despite a satisfactory initial reduction and a closely fitting plaster cast. This applies particularly to fractures of the lower shaft of the radius with inferior radio ulnar dislocation, fractures of both forearm bones and fractures of the shaft of the tibia. These fractures should be X rayed every second or third week during the first two months.

In the treatment of fractures economy of X ray films is fatal. It has been said that bones are filled not with red marrow but with black ingratitude. This is not true of the controlled treatment of fractures but if a surgeon neglects this letailed control black ingratitude is his just reward.

Plaster-of-Paris Technique

Thousands of years ago the Egyptians immobilised fractures by means of linen stiffened with gum or plaster. Starch, clay and egg albumin were subsequently employed. In the last century walking plasters were used by Krause in the treatment of leg fractures and in 1887 he reported a successful series of ninety eight cases. He fitted a special laced boot over the plaster for walking out of doors. Unpadded plasters and U shaped



FIG. 294

Preparing plaster bandages

walking iron stirrups were employed by Korsch in 1894.¹ More recently the unpadded plaster has been popularised by Böhler and his associates.

Preparation of plaster bandages—The best dental plaster should be used and a fairly wide meshed starch free crinoline or muslin bandage 6 in wide and 5 yds long. The plaster is firmly and evenly rubbed into the crinoline which is lightly rolled (Fig 294). If the rolling is too loose the central core slides out during application. If it is too tight the bandage will not soak evenly. The completed plaster bandage should be elastic and springy. The proportion of plaster to crinoline depends on individual preference but too much crinoline makes an expensive bandage and too much plaster a brittle cast. An average 5 yd plaster bandage should weigh 8 oz and contain 85-90 per cent by weight of plaster. The Fracture Clinic which is regularly using plaster of Paris has no difficulty in preparing inexpensive bandages of perfect quality. In other circumstances one of the proprietary bandages such as Cellon² plaster should be used.

Plaster slab technique—The plaster bandage is lowered into a deep bowl of warm water and left completely immersed until bubbles cease to rise.

¹ Monroe. History of Plaster of Paris in the Treatment of Fractures. *Brit Jour Surg* 1930; 6: xxiii-67.

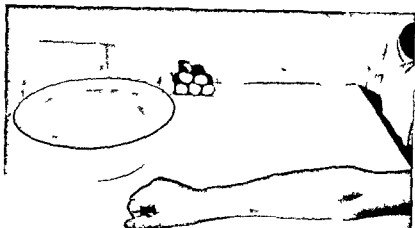


FIG 295

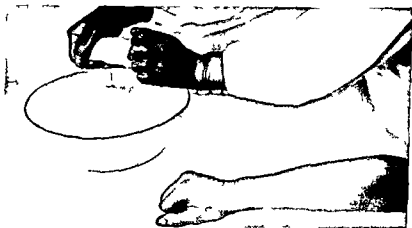


FIG 296



FIG 297

Plaster slab technique. The bandage is soaked until bubbles cease to rise. While holding each end it is lightly squeezed. The wet slab is rapidly prepared.

It is then lightly squeezed from the two ends. A slab of the required length and width is prepared on a glass or enamelled surface (Figs 297-299). A proper cast is not a series of discrete layers of plaster but one homogeneous mass. The bandage must therefore be sufficiently wet and sloppy, each layer must be firmly rubbed in, and the work must be completed rapidly. While the plaster is still quite wet and before it has had sufficient time to



FIG 298

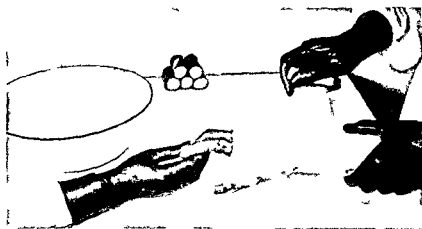


FIG 299

Plaster slab technique. The plaster is moulded to the contour of the limb and held in position with wet open weave bandage.

set it is applied to the limb, firmly moulded round the bony prominences and bound lightly in position with a wet open mesh bandage. Movement of the joints during setting of the plaster will produce ridges at the joint level, sometimes with disastrous results (see p 179). This must be avoided by keeping the limb immobile until the plaster is hard.

To immobilise a wrist, the slab is made of sufficient length to allow it to be turned back double over the hand and wrist. Similarly, an elbow may be immobilised by a plaster slab down the back of the limb. For an ankle more than one slab may be used. One is applied from the upper

calf over the heel and sole to the toes, and a second longer slab down one side of the limb, under the heel and up the other side

Preparation of dry slabs—Time is saved and the danger of applying a half set plaster to the limb is avoided if dry slabs are ready prepared (Fig 300) The crinoline into which the plaster is rubbed is not rolled in the ordinary way, but is folded on itself backwards and forwards to produce not less than six layers Any length may be prepared, it is convenient to use 18 in by 5 in slabs for the upper limb and 30 in by 6 in slabs for the lower limb The completed slab is folded, ends to the middle, so that it can be stored and subsequently soaked exactly like a rolled plaster bandage It is then rapidly opened out and immediately applied to the limb

Complete plaster cast—A complete cast is constructed by applying one or more slabs, carefully moulding them in position with a wet soft bandage, and then applying ordinary plaster bandage in a circular manner *These encircling turns of plaster bandage must not be pulled tightly, they are laid on the limb without tension* When necessary a slight pleat is taken in one margin of the bandage in order to preserve an even and smooth application Each layer is firmly rubbed in and the plaster while still wet is moulded round bony prominences to conform exactly with the contour of the limb This moulding is done with the palm and then *an eminence rather than with the finger tips*

Padded or unpadded plasters?—When a limb is put in plaster shortly after injury and before it has begun to swell, padding should always be used A double faced wool bandage can be applied smoothly and evenly (Fig 301) Similarly, after operations performed with a tourniquet, operative excision of wounds or compound fractures sequestrectomy, or any other operation which will cause reactionary swelling, the plaster should be padded On the other hand, when the plaster is changed after two or three weeks and it is known that there will be no further swelling, it is better to use an unpadded cast *The plaster must fit very closely it may be applied directly to the skin, or over a layer of stockinet* Bony prominences, particularly the neck of the fibula the iliac spines, and the spinous processes, should be protected with $\frac{1}{2}$ in adhesive felt Friction at the upper and lower margins of the cast should be prevented by a strip of felt or wool

Reinforcement of plaster—The best protection against breaking is a completely uniform plaster of even thickness at all levels Occasionally a patient may break plasters so rapidly that it is useful to incorporate a layer of aluminium wire mesh across the joint level A double hip spica is best reinforced by joining the two thigh casts with a "rope" of plaster bandage as a cross strut Hip spicas may also be reinforced by "ridging" Plaster slabs applied across the joint level are pinched together down their whole length to make a series of vertical ridges

Wedging of plasters—It is of the utmost value to be able to correct minor degrees of angulation without completely changing the plaster If the plaster is cut down and a new one applied, some other displacement may arise The surgeon is tempted to "leave well alone" Yet the slightest degree of angulation should be corrected, and a simple device is available by which this can be done with absolute control

A linear cut is made round two thirds of the plaster at the level of fracture on the concave side of the angle A short gas anæsthetic may be



FIG 300

Preparing dry plaster slabs. The ends are twice folded to the middle. The slab is then conveniently stored and soaked like an ordinary bandage.

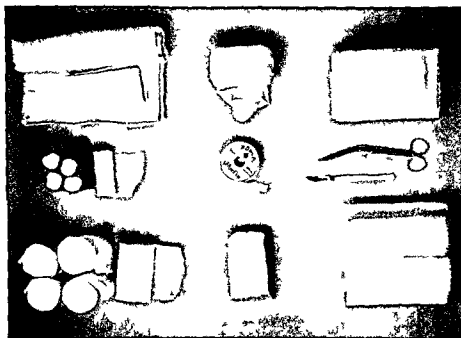


FIG 301

Materials required for application of plaster. Adhesive felt, stockinet and plaster slabs, open weave bandage, adhesive strapping, scalpel and bandage scissors, rolled double faced wool bandages, sorbo rubber and plaster bandages.

given and the linear division is opened to a wedge which is held open by a small block of wood placed between the two cut edges (Figs 302-304). The block must be exactly opposite the angle in front of the limb for backward angulation, on the inner side for outward angulation, half way between the two for combined backward and outward angulation and so on. Another radiograph is then taken and the degree of correction may be increased or decreased by inserting larger or smaller blocks of wood. When

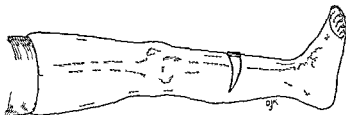
FIG 302



FIG 303



FIG 304



Wedging a plaster to correct angulation. A linear cut is opened to a wedge held by a wood block, and after confirming the position by X-ray the gap is repaired with plaster. (By courtesy of *Four Bone and Joint Surgery* from a hospital article 1935 x 591)

a final radiograph proves that the alignment is perfect the gap in the plaster is filled and reinforced.

This procedure is invaluable in the treatment of fractures of the tibia and fibula. It allows perfect control and accurate correction of the slight degrees of angulation which cannot always be avoided with the initial plaster. It may also be used in fractures of the shafts of the forearm bones and in trochanteric fractures of the femur treated in a hip spica (Figs 302, 304). Caution is necessary if it is proposed to correct a severe degree of angulation. Gross wedging may so increase the pressure of the plaster on the limb even a considerable distance above and below the level of the wedge that pressure sores may develop unless the possibility is borne in mind and discomfort relieved by cutting windows.

Walking plasters—A walking plaster for the lower limb should be

reinforced with an extra slab beneath the sole extending to just above the back of the ankle. Irregularities are made good and a flat walking surface produced. The patient should not walk until the plaster is thoroughly dry. The impact of weight bearing may be reduced by applying a $\frac{1}{4}$ or $\frac{1}{2}$ in. heel of sorbo rubber. This is held in position by a few turns of strapping or Elastoplast. A plaster boot is made with a leather sole so that the patient can walk out of doors even in wet weather and appear normally dressed (Figs 307-308).

Walking iron stirrups projecting beyond the heel are often incorporated in the plaster. Although they reduce wearing of the plaster most patients



FIG 30



FIG 30b

Wedge placed to increase the abduction of a trochanteric fracture
Radiographic control is used

acquire a bad gait. Either they walk rigidly as if on a stilt with the knee stiff or they pivot on the heel twisting the whole limb out with each step. It is very much easier to achieve a normal heel and toe movement with an ordinary plaster with or without a sorbo rubber heel. Moreover it is difficult to fit a boot over a walking iron stirrup and a closely fitting plaster boot adds a great deal to the patient's self respect. It enables him to walk in streets and shops almost unnoticed. Many patients have learned to pursue a normal life despite a below knee walking plaster cycling driving riding playing golf and even hunting and deer stalking.

Removal of plasters—A professor of surgery retains one vivid childhood memory—the agony of removal of a plaster. The pain of the fracture is forgotten but not the pain of cutting down the plaster. Even the most gentle of surgeons seem to lose their sympathy on these occasions to brush aside the victim's protests and to hasten through the work as if time was the only factor. One patient's thigh was completely split open from top to

bottom by the cutting edge of a plaster shears wielded by a vigorous young house surgeon. The patient was under an anæsthetic but forty stitches in a 14 in wound were not easily explained.

The first essential is not to frighten the patient and to realise that even the sight of plaster shears may terrify a child. The second is to choose good shears. The blade which is inserted beneath the plaster should be slightly longer than the other so that it does not cut out with each bite but it must not be more than $\frac{1}{2}$ in longer and it must be shallow. Much of the pain and discomfort is due to the surgeon's struggles to push the deep blade in advance of the cut. The third precaution is to use the



FIG 307

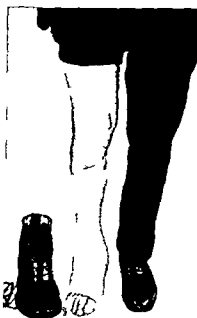


FIG 308

Walking plaster. A sorbo heel is incorporated, a plaster boot is worn and the patient dresses normally and pursues normal activities.

shears gently. The deep blade must be kept parallel with the skin and not pointed down at it. Short bites should be taken. Special care is necessary at joint levels where bones are prominent beneath the skin. Over these areas the plaster should be rubbed away a millimetre at a time.

Motor saws have been devised for cutting plasters but they have proved unsatisfactory. The instruments are difficult to use and they frighten the patient.

Protection from disuse œdema—As soon as a lower limb plaster has been removed an elastic support must be applied. The circulation has become accustomed to a rigid external support and when this is suddenly lost severe œdema develops. If it is left uncontrolled it may recur every day for many months or even for years. Every time the fluid leaks from the capillary field into the tissue spaces it opens up tracks which make it easier to leak again. The œdema causes the formation of adhesions (p. 154) and

the weight of the swollen limb interferes with the exercise which is necessary to promote venous return and restore the circulation. Massage is ineffective. It does no more than disperse the fluid temporarily.

There must be no interval between removal of the plaster and the application of elastic support. If in error an interval has been allowed and the limb is already swollen the patient must be put to bed for twenty-four hours with the foot elevated before the dressing is applied. Ordinary crepe bandages are useless because they slip and because the oedema is aggravated if the patient applies the upper turns of the bandage more tightly than the lower. An elastic stocking is more effective but it is expensive and liable to stretch. Elastic adhesive strapping often causes skin irritation and dermatitis.

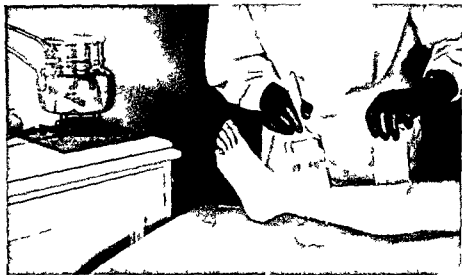


Fig. 309

Applying Unna's paste dressing to prevent oedema after removal of plaster

Unna's zinc gelatine paste is the most useful dressing. The cubes of gelatine are placed in a pot surrounded by water which is boiled until the gelatine melts to a smooth paste. It is then painted on the leg and covered with a soft bandage (Fig. 309). Two or three alternate layers of paste and bandage complete the dressing which must extend from the webs of the toes to just below the knee. Proprietary bandages already impregnated with a gelatine paste may be used. Viscopaste bandage requires no preparation but dries with a slightly harsh surface. Ichthopaste bandage requires immersion in hot water for ten or fifteen minutes and at first smells of ichthyol but it is non-irritant and soothing to a tender skin.

The dressing must be retained until the muscle tone and circulation of the limb are restored. The patient's observation that the dressing is no tighter at the end of the day than it was at the beginning shows that the tendency to oedema has subsided. In the adult protection is necessary for five or six weeks and sometimes for several months.

Complications of Plaster Immobilisation

Gangrene arising after a fracture is usually due to arterial injury and it may be an unavoidable complication (p 127). In recent years there have been one or two tragic cases of gangrene where the pressure of an unpadded plaster has been the most important if not the only cause. The circulation must be tested frequently. It is not enough to compress the tips of fingers or toes and confirm that the anæmic area refills with blood. Fluid blood often remains in the digit and the sign may be observed even after complete stasis of the circulation. The anæmic area must flush rapidly with blood; the digits must be warm; they must not be cyanosed or pale; they must be pink. *If the return of blood after compression of a digit is slow the patient must not be allowed out of sight.* The test must be repeated every ten or fifteen minutes. If the digit is blue or cold and pallid the plaster must be cut at once. It is usually sufficient to make a longitudinal cut through the whole length of the plaster dividing every turn of wool or bandage until skin is exposed in the gap between the cut edges. The gap should then be filled with a light padding of wool. If necessary the whole of the front half of the plaster may be removed. If the pallor and anæmia are still unrelieved it is clear that the artery has been injured or that it is thrombosed.

Pressure sores may develop from the localised pressure of splints, bandage or plaster especially over bony prominences. Plaster sores may be due to

- (1) pulling one turn of the plaster bandage too tightly so that there is a ridge on the deep surface
- (2) careless moulding of the plaster causing undue pressure over a bony prominence
- (3) application of a plaster slab which instead of being wet and sloppy has begun to harden and does not adapt itself smoothly to the contour of the limb
- (4) movement of a joint during the setting of a plaster so that a ridge is formed
- (5) failure to protect bony prominences in a thin or emaciated patient
- (6) allowing an imperfectly set plaster to rest on a hard surface so that it is flattened over a bony prominence (especially the back of the heel in leg plasters and the sacrum in hip and trunk plasters)
- (7) pushing combs, heads of knitting needles, small wads of wool or other foreign bodies between the plaster and the limb
- (8) delay in repairing a crack in the plaster near a joint so that there is friction of the skin against the broken margins

Signs—The patient may complain of persistent localised discomfort or pain. These symptoms should never be ignored even if they pass off in a day or two because the tissues rapidly become anæsthetic. In some cases there has never been any complaint of pain and the first sign may be the typical smell of accumulated secretions and discharge. The sore can easily be localised because the overlying plaster becomes much hotter than elsewhere. Oedema of the toes or fingers recurring after the initial oedema has subsided means almost certainly that there is a pressure sore. If the sore is near the end of the plaster the digits become red and inflamed or

dusky. Finally the sloughing is recognised by the staining of the overlying plaster or by a purulent discharge from the end of the plaster.

Treatment—As soon as a pressure sore is suspected a small window must be cut. Even if the suspicions were unfounded no harm has been done and the patient must not be allowed to feel that he has complained unnecessarily. Whether there is a sore or not the window must be filled with a pad of wool firmly bandaged into position. Unless this is done there will be oedema of the unprotected area which will aggravate a threatening ulceration or even produce new sores round the margin of the window. If there is already ulceration a moist dressing is applied until sloughs have separated and then a dry or vaseline gauze dressing which is not changed more than once a week.

Oedema distal to the plaster—Oedema of the fingers and toes is inevitable after injuries of the wrist and ankle especially when local swelling is prevented by a plaster cast. This reactionary swelling is of little significance but the limb should be elevated until it has subsided. Active exercises of the swollen digits are even more important than usual. It is a mistake to try to prevent swelling of the toes by cutting the lower



FIG. 311

Paste dressing, crepe bandages and plasters must extend to the web of the toes otherwise blistering and gravitational oedema develop in the unprotected forefoot and there may be ulceration at the margin of the dressing.



FIG. 310

A plaster sore

The patient's complaints of minor pain and discomfort were more real because he was a querulous difficult type who was always complaining. Even minor complaints must always be accepted; a window must be cut or the plaster be valved.

This reactionary swelling of the toes by cutting the lower edge of the plaster back to a more proximal level. In the lower limb most of the oedema is gravitational and the more the plaster is removed the greater the area which will swell and the more inevitable is pressure friction and ulceration of the skin at the margin of the plaster (Fig. 311). For the same reason a plaster spica or a plaster for the knee should never finish in the lower leg leaving the foot and ankle unprotected. If the ankle is not to be immobilised a viscopaste bandage must be applied to the foot from the webs of the toes and incorporated in the lower margin of the plaster.

Purulent dermatitis—As a rule when plaster is applied directly to a limb without padding the skin becomes dry and scaly. Some patients have a more susceptible skin and a dermatitis develops similar to intertrigo dermatitis. Staphylococcal infection of the hair follicles and sweat glands

supervenes and if the condition is ignored a severe and extensive purulent dermatitis follows. The first sign is itching and irritation followed later by an intolerable burning pain. At the first sign of irritation a small window should be cut and talcum powder applied and blown under the margins of the plaster every day. It is sometimes necessary to apply a new plaster over a double layer of well powdered stockinet.

Skin blistering is common during the first twenty four hours after severe injuries of the ankle, leg and elbow. The blisters are due to traumatic œdema of the cuticle and the exudate is sometimes hæmorrhagic. They only develop where the skin is unsupported. If splints are used they appear between the splints not beneath them. They never develop beneath an encircling plaster. If the skin is already blistered the blebs should be emptied by pinching a minute hole in the overlying cuticle. Talcum powder is dusted over them and plaster applied in the usual way.

General Complications

Hypostatic pneumonia—When an elderly patient sustains an injury necessitating recumbency there is a tendency to hypostatic congestion of the lungs leading sometimes to pneumonia. This tendency is increased if the patient is unable to turn unaided owing to a heavy plaster cast over the trunk and hips. Regular turning from one side to the other and when possible on to the face should be carried out every few hours during the day. As soon as possible the patient should be propped up with pillows.

Renal calculus formation—If recumbency is necessary for several months it must be borne in mind that there will be a generalised disuse decalcification of the whole skeleton with a greatly increased output of calcium salts by the kidneys. To the generalised decalcification of all bones may be added the more marked local decalcification of the fractured bone particularly in the case of infected compound injuries. The high concentration of calcium salts in the urine may be responsible for extensive calculus formation (Fig 312). At first the calculi are soluble and they may disappear when the saturation of calcium salts in the urine is reduced by increased fluid intake and the renal tract drainage is improved by posture. Meanwhile however, there is a risk of renal inefficiency due to obstruction and the excessive excretion of calcium may itself lead to renal cellular degeneration. Moreover the deposit of calcium salts may provide a nucleus for the formation of insoluble calculi. In some cases pyonephrosis has led to loss of a kidney or even to death from uræmia.

Particular caution is necessary in compound fractures of the lower limb, in fractures of the hip, in injuries of the pelvis with rupture of the bladder or urethra and in spinal injuries treated in recumbency. Postural drainage of the kidneys must be encouraged by raising the head end of the bed on blocks for several hours each day. The fluid intake should be increased and regular exercises must be practised for those parts which do not need to be immobilised. In order to render the urine acid and so maintain solution of the urinary calcium phosphate, a diet yielding an

acid ash is recommended. Details of an acidogenic diet are given by Pyrah and Fowweather.¹



FIG. 312

Nephrolithiasis due to recumbency in a case of fracture dislocation at the eleventh and twelfth dorsal level.

Fat embolism—Of the three complications of fractures which may cause sudden death shock is usually fatal at about the third hour fat embolism on the third day and pulmonary embolism during the third week. Fat embolism though seldom recognised clinically is the cause of death in over 20 per cent of fatal fracture cases.^{2,3} It has been assumed that the fat globules gain entrance to the circulation from the medulla of the injured bone through the ruptured walls of blood vessels but it is also possible that the emboli may consist of the fat of blood plasma broken down from its normal state of emulsification by histamine or other products of muscle injury or by ether inhaled during anaesthesia.^{4,5} The emboli lodge in the lungs or brain or are scattered throughout the body. The pulmonary

¹ Pyrah and Fowweather "Urinary Calculi in Recumbent Patients," *Brit. Jour. Surg.* 1938, xvi, 93.

² R. A. Rowlands and C. I. G. Wakeley "Fat Embolism (full cinematography)," *Lancet* 1941 i, 4.

³ Vance *Arch. of Surg.* 1931 xxi, 4 and *Amer. Jour. Surg.* 1934 xxvi, 27.

⁴ Watson "Fat Embolism," *Brit. Jour. Surg.* 1936-37 xxiv, 670.

⁵ Lehmann and Moore *Arch. of Surg.* 1927 xiv, 671.

type sometimes develops within a few hours of injury and clinically resembles acute oedema of the lungs but more often dyspnoea cyanosis precordial pain cough and hyperpyrexia develop gradually during the first few days and simulate broncho pneumonia In the cerebral type the symptoms are those of delirium tremens and cerebral irritation passes slowly into stupor coma and death The diagnosis may be confirmed by the presence of fat in the urine and sputum¹ When the onset is delayed and pulmonary signs predominate the prognosis is good but in an established case of systemic embolism with cerebral signs recovery is exceptional The only recognised treatment is that of prevention the gentle handling of fractures in first aid and subsequent treatment the institution of complete immobility at the first possible moment and the use of tourniquets for all bone and fracture operations²

MANIPULATIVE REDUCTION AND CONTINUOUS TRACTION

Two strong men will suffice by making extension and counter extension —Hippocrates 350 B.C.

Principles of traction—When the shaft of a long bone is fractured the elastic retraction of muscles surrounding the bone tends to produce overriding of the fragments This tendency is greater (1) when the muscles are powerful and long bellied as in the thigh (2) when the fracture is imperfectly immobilised so that there is pain and therefore muscle spasm (3) when the fracture is mechanically unstable because the fragments are not in apposition or because the fracture line is oblique

Upper limb—In injuries of the upper limb the first and second aggravating factors can be excluded The muscles are not particularly



FIG. 313

Traction in the upper limb is required only for unstable fractures such as Bennett's fracture-dislocation of the thumb

strong and long bellied There is no difficulty in securing complete immobility and therefore in relieving muscle spasm Continuous traction to neutralise the elastic recoil of muscles is required therefore only in fractures which are structurally unstable It is necessary in Bennett's fracture dislocation of the thumb metacarpal No matter how perfectly the fracture is reduced and no matter how valiant the efforts to hold it reduced the obliquity of the fracture line is such that it has no structural stability

J. I. Scott F.R.C.S. & H. Kenyon A. Robt-Scott Fat F. bolson, Sput n. F. ca. Inat! Lancet 1910 1 11
Ryerson J. J. Am. Med. Assoc. 1910 11 11 6

There is no opposition to the retraction of the long flexor and extensor muscles. If a perfect result is to be achieved manipulative reduction and immobilisation must be supplemented by continuous traction for the first three weeks (Fig. 313). There are one or two other unstable fractures of the upper limb



FIG. 314

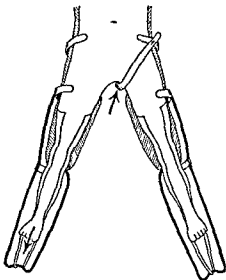


FIG. 315



FIG. 316

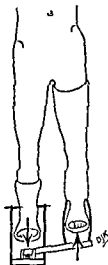


FIG. 317

Fixed types of lower limb traction. The counter pressure is against the groin in the Thomas splint (Fig. 314) the opposite groin in the abduction frame (Fig. 315) and the sole of the opposite foot in well leg traction plasters and splints (Figs. 316 317)

which sometimes require continuous traction (phalanges, Chapter XXVII, shaft of radius Chapter XXV neck of humerus, Chapter XXII)

Lower limb—In the lower limb, the power of the muscles and the range of retraction is greater and there may be more difficulty in achieving perfect

immobility Almost every fracture of the shaft of the femur requires traction throughout the period of immobilisation, and unstable fractures of the leg bones may need traction for five or six weeks

Principles of counter-traction—A team practising tug of war must either fix their rope to a wall or they must pull against another equally strong team. Similarly, traction on a limb requires a fixed point from which the traction is exerted, or an equal counter traction in the opposite direction. Each of these methods is employed. The first is known as fixed traction and the second as balanced traction.

Fixed traction—With a Thomas' bed splint, traction is exerted from the fixed point of the patient's pelvis. The extension tapes pull the limb down to the splint, which is prevented from moving in the opposite direction by the resistance of the ring of the splint against the tuber ischi (Fig 314). With an abduction frame, traction is exerted from the fixed point of the groin strap on the opposite side (Fig 315). "Well leg traction" is exerted from the fixed point of the sole of the opposite foot.¹ This method was devised to avoid the pressure of the ring of a Thomas' splint in the groin. The opposite limb is put in a plaster spica and the bars of the splint are incorporated in it (Fig 316). The counter pressure is then transferred from the groin to the plaster spica and therefore to the sole of the opposite foot. This principle is used in the Roger Anderson splint for certain fractures of the upper end of the femur. Although with this splint the plaster on the normal limb is not a complete spica, the counter pressure is transferred to the opposite sole in a similar way (Fig 317).

Balanced traction—The simplest type of balanced traction is that used in weight extension. A weight is hung over a pulley at the foot of the bed, and fixed to the limb by extension strapping on the skin or by a traction pin through the bone. The counter traction is the patient's own body weight sliding down the bed which is raised at the foot (Fig 318). The 10 or 15 lb weight on one side of the pulley is balanced by the patient's own body weight on the other. This principle is used in Braun's splint for fractures of the femur and tibia and in Hodgen's splint or any other splint which is suspended from an overhead beam.

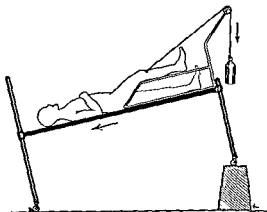


FIG 318

Balanced traction. The traction of the weight on one side of the pulley is balanced by the counter traction of body weight on the other.

Combined fixed and balanced traction—Most surgeons who use the Thomas' bed splint regularly, use combined fixed and balanced traction. The splint is applied in the ordinary way with the ring firmly pressed against the pelvis and the extension tapes tightened. As the traction continues the extension tapes tend to slide and to become slack, making it necessary to tighten them several

¹ Roger Anderson. A New Method of Treating Fractures Utilising the Well Leg & Counter traction. *Surgeon General's Journal*, 1934, 54, 70.

times a day. Furthermore, the pressure of the ring of the splint causes discomfort or even pressure sores in the adductor region and groin. Both these difficulties are met by fastening the end of the splint to the foot of the bed which is raised 12 or 18 in. The patient is then partly suspended from the foot of the bed by the extension tapes. Some degree of balanced traction has been added. The patient tends to fall away from the ring of the splint and pressure in the groin is reduced. Moreover slack in the extension tapes is at once taken up, and they need tightening only once a day or on alternate days.

Similarly, with an abduction frame the efficacy of the traction can be increased, and the discomfort of the groin strap relieved by fastening the frame to the raised foot of the bed.

Sliding bed traction—Pugh first utilised the simple device of applying extension tapes to the limb and fastening the tapes to the raised foot of the bed to secure traction on the hip joint. If only one limb is fastened in this way the opposite side of the pelvis slides farther down the bed than the fixed side, and the hip is slowly abducted. The device is useful in treating epiphyseal coxa vara and avascular necrosis of the upper femoral epiphysis, where traction and abduction are required but not immobilisation. The extent to which the patient slides down is reduced by friction against the mattress and bed clothes. Hendry has excluded this friction in a "sliding bed". The mattress is placed on a wooden surface which slides on roller bearings over the bed itself (Fig 319). The amount of traction which requires a tilt of 30° or 40° on an ordinary bed can be achieved on a sliding bed with only a 10° or 15° tilt. The available traction is almost unlimited and the patient's comfort is greatly increased. Actually the patient's head need not be any lower than his feet not even 10° or 15°. Provided that the bed itself is raised at the foot the false top will still slide down and give the necessary traction whether it is parallel with the bed or not, and in the latest model of Hendry bed the sliding top is horizontal, although the bed on which it slides is tilted.

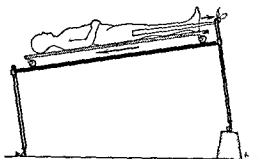


FIG 319

Sliding bed traction (Hendry and Naughton Dunn). By excluding friction a 10° tilt on a sliding bed gives as powerful traction as a 40° tilt on an ordinary bed.

Dangers of traction and distraction—A clear distinction must be drawn between the heavy traction which may be applied for a few minutes as part of the manoeuvre of manipulative reduction of a fracture, and the lighter traction which is maintained continuously in order to prevent redisplacement. Continuous traction must not be used for the reduction of a fracture. It is a mistake, for example, to treat a fracture of the shaft of the femur without initial manipulation but simply by the suspension of a weight from a skeletal traction pin in the bone with the object of correcting displacement gradually over a period of days or weeks. Two or three weeks may elapse before it is recognised that reduction is imperfect, the weights may then be increased and there is danger of distraction, repeated adjustments are made and there is constant interruption, after long delay it may be found that operative

reduction is necessary. Such a routine is very harmful to the healing fracture. It disturbs the growth of granulation tissue and callus exactly at the time that freedom from disturbance is most important. Frequent interruption, alteration of position, distraction of fragments and late operative intervention are all factors which seriously delay union.¹ The purpose of treatment of any fracture should be to complete the final reduction of displacement within two or three days and then to maintain the reduced position continuously and without interruption. In the case of a fractured femur displacement must be reduced by manipulation and if necessary heavy traction under anaesthesia, the limb then being immobilised in splints with just sufficient continuous traction to prevent redisplacement. Heavy continuous traction is avoided, and under no circumstances must the fragments be distracted (pp 18-22). Similarly, fractures of the tibia must not be over pulled. If a fracture is unstable and continuous traction is needed not more than about 10 lbs should be used. Fractures which would otherwise unite in eight or ten weeks will, after distraction, unite only in eight or ten months. Such a delay is entirely unjustifiable. It would be much better to control the instability and prevent redisplacement by internal fixation.

MECHANICAL REDUCTION AND SKELETAL TRANSFIXION

This is a mechanical age and mechanisation has invaded the realm of fracture treatment. A technique has been developed in America of transfixing both the fragments of a fractured bone with steel pins, fixing the pins in the stirrups of a machine, and then reducing the fracture under X ray or fluoroscopic control by mechanical traction, angulation and rotation. When reduction is complete the projecting ends of the pins are incorporated in a plaster cast (Figs 320-321). Alternatively the pins may be fixed in the clamps of a steel bar which acts as an extra skeletal "bone" bridging the fractured bone and providing all the fixation required (Fig 288). The object of such treatment is to avoid the necessity for plaster or splints and allow early mobilisation of the joints. A single pin in each fragment is inadequate for this purpose because the pin can rotate in its track, and the fragment can rotate round the pin. At least four pins must be used, two in each fragment. Furthermore, if the pins are parallel to each other, movement of the fragments may still occur (Haines' technique Figs 289-290). A two pin unit must consist of pins driven into the bone at an angle of 45° (Roger Anderson technique).

These methods of skeletal transfixion and machine reduction are of doubtful merit, moreover, they are fraught with danger. The main advantage which has been claimed is that early mobilisation of joints is possible. But in fractures of the tibia there is no serious problem of stiffness of the knee joint whatever method of treatment is used, and in fractures of the femur where there is a serious problem of joint stiffness, the range of movement which is possible in the knee joint after transfixion of the quadriceps muscle at four levels is very limited indeed. The disadvantages of the technique are (1) except in the skilful hands of Roger Anderson himself, there is danger of distraction of the fragments and delayed union, or of inadequate immobilisation of the fragments and non union, (2) in the hands of every surgeon,

¹ R. Watson-Jones and W. D. Collett. New Union of Fractures. *Brit Jour Surg* Jan 1917

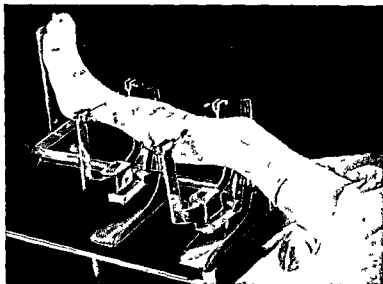


FIG 320

Simple type of mechanical reduction apparatus for fractures of the shafts of long bones. In this particular machine a single transfixion pin or Kirschner wire is driven through each fragment and then incorporated in stirrups, which by mechanical means can be distracted, angulated or rotated in relation to each other. (Reduction by Wing Commander A. Puller.)

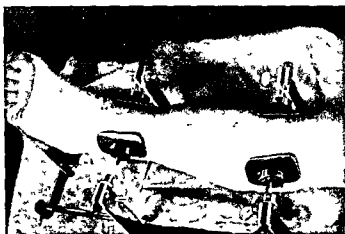


FIG 321

The Kirschner wires are incorporated in the plaster, and held taut by button tighteners on the surface of the plaster, the projecting ends are cut off. It is to be noted that a single pin in each fragment does not arrest movement, and angulation can recur despite pins and plaster. Four pins are necessary, two in each fragment, at an angle of 45° to each other. The merit of this technique is not yet proved.

reduction is necessary. Such a routine is very harmful to the healing fracture. It disturbs the growth of granulation tissue and callus exactly at the time that freedom from disturbance is most important. Frequent interruption, alteration of position, distraction of fragments and late operative intervention are all factors which seriously delay union.¹ The purpose of treatment of any fracture should be to complete the final reduction of displacement within two or three days and then to maintain the reduced position continuously and without interruption. In the case of a fractured femur displacement must be reduced by manipulation and if necessary heavy traction under anaesthesia, the limb then being immobilised in splints with just sufficient continuous traction to prevent redisplacement. Heavy continuous traction is avoided and under no circumstances must the fragments be distracted (pp 18-22). Similarly fractures of the tibia must not be over-pulled. If a fracture is unstable and continuous traction is needed not more than about 10 lbs should be used. Fractures which would otherwise unite in eight or ten weeks will after distraction unite only in eight or ten months. Such a delay is entirely unjustifiable. It would be much better to control the instability and prevent redisplacement by internal fixation.

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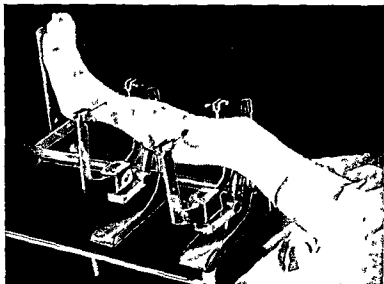


FIG 320

Simple type of mechanical reduction apparatus for fractures of the shafts of long bones. In this particular machine a single transfixion pin or Kirschner wire is driven through each fragment and then incorporated in stirrups which by mechanical means can be distracted, angulated or rotated in relation to each other. (Reprinted by permission of the Committee for the Reduction of Fractures.)



FIG 321

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no matter how skilled there is danger of infection of the pin tracks. Precautions can of course be taken to avoid gross infection but some degree of low grade infection of the pin tracks is inevitable it must develop sooner or later. In certain regions a low grade infection is unimportant but in other regions it may cause serious joint stiffness (p 58). If the treatment of a fracture necessitates perforation of the skin at all it is surely better to make a proper incision use internal fixation and seal the incision under strict aseptic control rather than to leave a continuous track occupied by a foreign body passing from the outside air through skin and soft tissue to bone. The method has not been received with favour in this country. Many of the traction machines which were distributed at the outbreak of war have been recalled and others are now accumulating dust in dark cupboards.

OPERATIVE REDUCTION AND INTERNAL FIXATION

The most unfortunate innovation in the treatment of recent fractures is routine exposure and reduction by open operation. Thousands of human lives have been sacrificed and many more have been crippled — LORENZ BOHLER¹

The contentions of Lane are sound and have proved beyond question that when indicated and well done the operative treatment of fractures has given the best results — O NEILL SHERMAN²

Here are two diametrically opposed views. Where is the truth? The truth is in the words *when indicated and well done*. The surgeon who operates before he learns the principles of fracture treatment is responsible for crippling. The surgeon who has not even learnt to operate may be responsible for the loss of lives. An experience of the non-operative methods by which perfect results can be secured in the great majority of fractures is essential. With this knowledge and with an aseptic technique which can be relied upon the operative reduction of certain fractures is justified. On the other hand the surgeon who plates a fractured femur by an indifferent technique and then puts the patient to bed with the limb merely supported on a pillow (Fig 322) is not capable of treating such a fracture by any method much less by operative methods.

Disadvantages of operative reduction—To infect a simple fracture by a careless operation is a tragedy. The most scrupulous aseptic technique is necessary. The standards of asepsis which may be safe for abdominal surgery are unsafe for operations on bones and joints. Forty eight hour skin preparation ten minute scrubbing up complete screening of skin by stockinet or towels no touch technique and absolute integrity of the whole theatre staff are the elements of this technique. It is said that the safest period is the tenth to the fourteenth day but there can be no question of relative safety or of degrees of risk. With perfect precautions there should be absolute safety no risk and any day a safe day.

Apart from the possibility of infection there are other disadvantages. The stripping of muscles and periosteum which interferes with the blood

¹ Lorenz Böhler "Treatment of Fractures" John Wright Bristol 1935 8°
² O'Neill Sherman "Four Results in the Treatment of Fractures" *Trans Orthop. Surg. Amer Med Assoc.*, 1922.

supply of the bones and the dissemination and dilution of the fracture hæmatoma may delay repair. Furthermore exposure of the bone may promote intermuscular adhesion formation and joint stiffness. This applies particularly to fractures of the femoral shaft. Whatever exposure is used fixation of the quadriceps muscle is difficult to avoid and sometimes the recovery of knee movement is delayed or imperfect.



FIG. 300

The result of operative reduction of a fracture of the shaft of the femur by a surgeon who was untrained in the principles of fracture treatment. The operation was badly done and the patient then lay in bed with the limb on a pillow. There is gross angulation of the femur, complete anchorage of the quadriceps, 90° drop foot and nail clanging of the toes.

Optimum time for operative reduction.—The decision that a fracture requires treatment by manipulation and plaster manipulation and continuous traction or operative reduction and internal fixation should be made within a few days of injury. It is not justifiable to try first one method and some days later another after several weeks a third and finally adopt operative reduction as a last resort. The principle of timing of fracture treatment must be respected. There should be no interference with the healing process during the vital period from the second to the tenth weeks. As a rule the decision can be made when the fracture is first examined and if an operation is required it is performed at the optimum time within a few days of injury. Even if it is decided that one or more manipulations should be tried first these manipulations should be completed and the decision made within three or four days.

Indications for operative reduction.—There are three main indications for operative reduction and internal fixation —

- (1) The probability that manipulative reduction will not succeed or the fact that it has been tried and has failed
- (2) the probability that manipulative reduction will not be maintained or the fact that redisplacement has actually occurred
- (3) the probability that union will be slow

INDICATIONS FOR OPERATIVE REDUCTION

1 **Manipulation will not succeed**—The failure of manipulative reduction may be due to (a) displacement of fragments by elastic retraction of muscles (b) displacement of fragments with interposition of soft tissues (c) displacement of fragments still unreduced many weeks or months after injury or (d) the displacement of small intra articular fragments which are not accessible to manipulation *Elastic retraction of muscles*—Fractures of the patella may be widely displaced by retraction of the quadriceps muscle or fragments of the olecranon by the triceps muscle. These fractures are to be regarded as bone injuries incidental to the rupture of tendons which necessitates operative suture. Other tendon ruptures or avulsions accompanied by fractures do not necessarily demand operative treatment. Fractures of the internal epicondyle of the humerus due to avulsion of the flexor muscles of the forearm are often treated successfully by simple flexion of the elbow, avulsion of the anterior iliac spines by avulsion of the sartorius and rectus femoris calls only for flexion of the hip. Fractures of the tuberosity of the humerus with retraction of the supraspinatus are treated by abduction of the shoulder. Fractures of the base of the fifth metatarsal by the pull of the peroneus brevis require simple immobilisation in plaster. It is because fractures of the olecranon and of the patella are also associated with interposition of soft tissues that operative reduction is usually necessary.

Interposition of soft tissues in fractures—Operative treatment is needed in fractures of the internal malleolus of the tibia with interposition of a flap of periosteum. Fractures of the shaft of the radius with interposition of the pronator teres tendon and fractures of the shaft of the femur with interposition of muscle. In fractures of the tibial malleolus the complication is shown by the radiographic evidence of a persistent gap between the fragments. In fractures of the shaft of the femur it is usually impossible to judge by radiographs alone that soft tissues are interposed. The important test is not only that manipulative reduction has failed but that it has proved impossible even with strong lateral pressure to bring the fragments into apposition and that throughout the manipulation no crepitus was elicited.

Interposition of soft tissues in dislocations—Joint dislocations may be irreducible by manipulation because soft tissue flaps are interposed. In dislocations of the elbow joint the tendinous origin of the flexor muscles may be drawn into the inner side of the joint together with a fragment of the internal epicondyle of the humerus which gives radiographic evidence of the complication. Manipulative reduction cannot be successful until the flap of tissue is hooked out. Similarly the reduction of dislocations of the knee joint may be obstructed by a flap consisting of the internal lateral ligament part of the quadriceps expansion and the capsule of the joint torn from the inner side of the femur and interposed between the articular surfaces of femur and tibia. Unlike the corresponding dislocation of the elbow joint there is no radiographic proof of the interposition but clinical evidence is available in the impossibility of replacing the bones in normal apposition no matter what strength is used and in the dimpling of skin over the inner side of the knee which occurs when strong lateral pressure is applied due to the pull of underlying tissues which are imprisoned in the joint. Metacarpal

phalangeal dislocations may also be irreducible by manipulation because the torn capsule of the joint is buttonholed round the metacarpal head.

Late unreduced fractures—When many weeks or months have elapsed since injury manipulative reduction may be impossible because the fragments are joined in malposition by scar tissue and callus. It is necessary to cut down on the fracture, freshen the bone ends, either angulate the fragments into position or secure reduction by strong traction and use internal fixation preferably by means of a bone graft.

Intra articular fractures—Small fragments may be chipped off the articular surface of a joint and necessitate excision—for example marginal fractures of the capitellum, the head of the radius and the femoral condyles. Similarly fractures of the tibial tuberosities may not always be amenable to manipulative replacement; it is sometimes necessary to open the joint and replace articular fragments.

2 *Manipulative reduction will not be maintained*—Certain fractures are unstable and prone to redisplacement. Oblique and spiral fractures of the tibia, despite a perfect initial reduction and careful immobilisation in plaster are liable to redisplacement. The two alternatives which are available are continuous traction and internal fixation. If the surgeon can be certain that his aseptic technique is completely reliable, open reduction and internal fixation is fully justified, particularly since methods of continuous traction often cause serious delay in the union of fractures of the tibia (see p. 19 and Chapter XXXV, Slow Union of Fractures). The same alternatives of continuous traction or internal fixation are available in fractures of the lower shaft of the radius with inferior radio ulnar dislocation (Fig. 330). In fractures of the upper shaft of the ulna with dislocation of the head of the radius (Monteggia fracture dislocation) the instability is best controlled by operative fixation of the fracture of the ulna. In fractures of the shafts of the forearm bones it is usually wise to try conservative measures first, but if the fractures cannot be perfectly reduced and the instability is therefore increased, there should be no hesitation in using internal fixation in order to prevent late redisplacement (Figs. 332-333).

3 *Union of the fracture will be slow*—The most striking example of a fracture which can be reduced perfectly but in which slow union justifies internal fixation is fracture of the neck of the femur. Most of these fractures would no doubt unite in time if carefully moulded plaster spicas were re-applied whenever they became loose and complete immobilisation was continued for the necessary period. But union is often so slow that the necessary period may be as long as twelve months or two years, and it is unreasonable to handicap an elderly patient in a plaster spica for so long a period. Internal fixation is obviously preferable. In other fractures the likelihood of slow union is recognised only three or four months after injury. Fractures of the lower shaft of the tibia and humerus may show little union despite complete immobilisation for several months. The fracture will certainly unite if immobilisation is continued long enough, but a bone-grafting operation may be performed as a time-saving measure. It is to be emphasised, however, that the repair of a slowly uniting fracture will not be accelerated by open reduction and plating. A plate is not an adequate treatment for slow union, delayed union or non-union; a bone graft is necessary.

SCRUPULOUS ASEPTIC TECHNIQUE

The general standard of aseptic technique in operating theatres is still far too low. It is a grave reproach that a post-operative infection rate of no less than 1 or 2 per cent has sometimes been accepted with complacency. The infection of one clean case in a thousand is a disaster of the first magnitude. Even slight delay in healing, redness of the skin or any other sign of wound reaction is evidence of failure. The ritual of asepsis can be learned only by training and experience. It must be acquired as a habit, subconscious, reflex, automatic, firmly ingrained in the minds of surgeon, assistant, theatre sister and nurse. All must share the responsibility and all must know they share it.¹ No single case of post-operative infection must pass unnoticed. A disaster has occurred and an inquiry should be held. Every detail of technique should be reviewed. A skilled watcher may be appointed for a period to draw attention to faults on the part of the surgeon himself or of any member of his team. Only then is the weight of responsibility fully realised.

Theatre—One theatre should be reserved for aseptic cases. Floors, walls, furniture and equipment must be phenolised daily. Windows must be heavily screened with gauze to prevent the entry of flies or of dust. Doors must be kept shut. No one may enter in outdoor clothes or footwear, neither on operating days nor on visiting days. Dust is the carrier of infection and every source of dust must be excluded. In ideal circumstances, the operating theatre should be air conditioned, all entering air being filtered and purified, the temperature is controlled in order to prevent the surgeon or his assistants from sweating. Ultra violet light may be used to sterilise the air.² It has been recommended that not only the air but also the theatre walls, ceilings and all equipment should be sterilised by liberating formaldehyde in the hermetically sealed theatre before putting the air conditioning plant in operation.³ These ideal conditions may not always be attainable but much more can be done to control dust borne infection than is now customary. We can stop all and sundry from walking in, we can stop tours of inspection by heavily booted officers and their entourage, we can stop the theatre from becoming the house surgeons' meeting place or the nurses' recreation room, we can bar entry to all who are not suitably clad.

Staff and visitors—Every individual who enters the theatre must be clothed in sterile gown, cap, mask and cloth boots, this applying at all times and not only when operations are in progress. Masks must cover the nose as well as the mouth and they must include cellophane sheets interposed between the gauze. Many apparently normal and healthy individuals are carriers of streptococci in the upper respiratory tract and the use of impervious masks is of the greatest importance. Talking in the theatre must be minimised, coughing and sneezing are prohibited. The cap should be deep enough to cover the eyebrows which often contain staphylococci. The junior theatre nurse must not enhance her charm by allowing stray curls to appear. The patient must not lie uncapped and unmasked as so often is the case and his coughing must be carefully controlled. Gowns must be closely tied at the back, no unsterile clothing must be visible. Nurses and visitors must stand back, they must never come in contact with instrument tables, theatre sister or surgeon.

Patient—The site of operation and a generous area above and below must be prepared repeatedly for not less than twenty-four hours and in the case

Prevention of Hospital Infection of Wound. Medical Research Council War Memorandum No. 6, 1931.
H.M. Stationery Office.

¹ L. Hart, *J. Hyg.* 1933, 1, 9.

² M. Gullin, *Ann. Surg.* 1914, 58, 67.

of foot operations for forty-eight hours. This long preparation is needed because two types of organisms exist in the skin—"transients" and "residents." Transient organisms are disposed of by a single washing with soap and water. Resident organisms are more securely dug in and they demand repeated washing, cleaning with ether and painting with antiseptics. The prepared area is covered with at least two layers of sterile towel. The outer layers are removed in the anteroom and the inner layer only when the anaesthetised patient is in position on the operating table. It is often unrecognised although it should be obvious that blankets and pillows carried from the ward teeming with bacteria must in no circumstances go into the theatre with the patient. The patient must be transferred in the anteroom from a ward trolley to a theatre trolley with specially sterilised sheets, blankets and pillows which are the equipment of the theatre and never leave the theatre.

Linen¹—Towels, side sheets, stockinet gowns, caps and masks are sterilised by steam under pressure. Sterile linen is taken from the tins by long handled sterile lifters, care being taken to avoid contact of the lifters or of the nurses' sleeve with the unsterile edge of the tin or its lid.

Operating sisters, assistants and surgeons—Surgeons and nurses must at all times avoid contact of their hands with infected matter.² Even before approaching the theatre their hands must be clean, their teeth clean and their hair clean. They must change into sterile clothes, caps, masks and phenolised rubber boots. For ten minutes by the clock they must wash fingers, hands and forearms, paying special attention to the sides of the nails, the webs of the fingers and the skin creases of the wrist. The hands and forearms are rinsed in antiseptic and dried on a sterile towel. A sterile gown is put on, taking care not to touch the outer surface of the gown or the sleeves. After the tapes have been tied by a nurse, a sterile back panel is fixed in position by the surgeon in such a way as to cover the tapes and the gap between the margins of the gown which have been contaminated by tying. This is important in lengthy bone grafting operations where in moving from one position to another the surgeon or sister may brush past an instrument tray. When fingers and hands have been washed for ten minutes and antiseptics have been applied, the skin is sterile, but it does not remain sterile. Within half an hour, resident bacteria previously in the pores are on the surface of the skin. Sterile gloves must therefore be worn. Even gloves do not afford full protection because about 20 per cent are punctured at the end of an operation and allowing for the hands of surgeon, assistants and nurses, there is at least one punctured glove in 75 per cent of operations. Weed and Groves³ recently found that one or more gloves had been perforated in 3,400 out of 4,540 consecutive operations. Infected sweat inside a rubber glove is under pressure and it is sprayed through any puncture hole there may be. One theatre epidemic was actually traced to a surgeon's hands carrying *Staphylococcus aureus*.⁴ For this reason,

¹ F. C. Cytler and others, Sterilisation and Aseptic Operating Room Technique, *Surg. Gyn. & Obst.* 1940, lxxi, 414.

W. Theilwall Thomas of Liverpool always carried a pair of rubber gloves in his hip pocket and wore them when examining an ulcer, a sinus or an infected wound. The gloves were then re-sterilised and returned to his hip pocket ready for the next occasion. He claimed that his hands were always sterile. Thomas responsible for medical training in Liverpool where he was influenced by Lister's teaching. He was largely responsible for introducing aseptic technique to Liverpool hospitals and with F. T. Paul of Liverpool the fame was a pioneer of abdominal surgery. He was the most dexterous surgeon I have known. Nearly every operation was completed without a single blood stain on the slide towels. This was his pride and any house surgeon who attended by allowing contact of a blood soaked drape with the towels never dared to offend again. One of the greatest privileges of my life was to be Theilwall Thomas's house surgeon—the last before his retirement.

² J. A. Weed and J. L. Groves, Surgical Gloves and Wound Infection, *Surg. Gyn. & Obst.* 1941, lxxi, 601.

³ E. A. Duvenhuyse and A. A. Miles, *Lancet* 1939, i, 1084.

even when sterile rubber gloves are worn special precautions must be taken in the handling of instruments the arrangement of the surgeon's instrument tray and the adoption of no touch technique

Instruments—All instruments must be boiled for not less than ten minutes and reboiled between every operation. The theatre sister must never touch an instrument by hand but only with sterile forceps or lifters. Needles are threaded with forceps and passed to the surgeon in a needle holder. Swabs are handed out with forceps. Instruments are sorted with forceps. No one in the theatre—nurse, sister, assistant or surgeon—must ever touch the business end of an instrument such as the blade of a retractor. If this should happen inadvertently or if any instrument touches the patient's skin it must be reboiled before use.

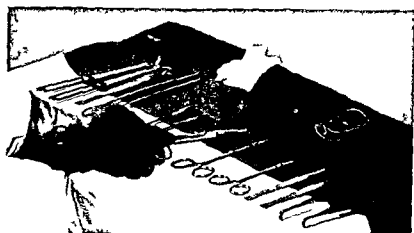


FIG 33

No-touch technique—surgeon's instrument tray

Clothes are often punctured during operations. The handles of instruments may therefore be contaminated; they must never come in contact with the business end of other instruments. The surgeon's tray is divided into two parts. The business ends of instruments are always replaced on the coloured cloth which is never touched by hand directly or indirectly.

Instrument tray—If instruments are left lying about or are placed haphazard on the surgeon's tray, the handle end of one instrument is likely to contaminate the business end of another. The surgeon's instrument tray should therefore be divided into two parts by means of a coloured cloth which is never touched by hand and never contaminated by contact with the handle end of an instrument. Immediately after use every instrument is replaced on the tray with its business end on the coloured cloth (Fig 323).

No touch technique—If it is necessary to palpate the site of operation before making the incision, contact with the patient's skin must be avoided by covering it with a sterile towel or gauze. As soon as the incision has been made the scalpel is discarded and not used again. The cut skin edges are painted with iodine and sterile side cloths are clipped over the margins of the skin. Bleeding points are secured with Spencer Wells forceps and if

In limb operations sterile stocknet may be used. The site of operation is painted with an antiseptic as usual. Sterile stocknet is pulled over the limb, unrolling it from foot to hip or hand to head. In such a way as to avoid contact of skin with the outer surface of the stocknet. The stocknet is then cut, making it unnecessary to use towels. It is then easy to hang up the limb without disturbing the towels or exposing the skin.

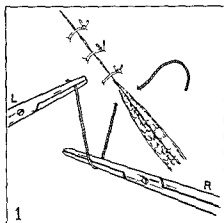


FIG. 324

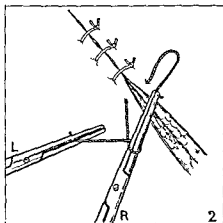


FIG. 325

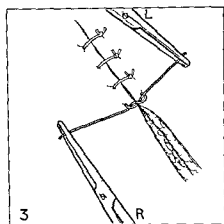


FIG. 326

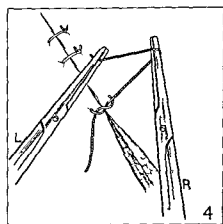


FIG. 327

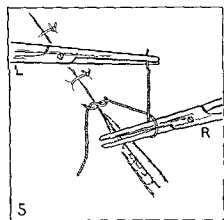


FIG. 328

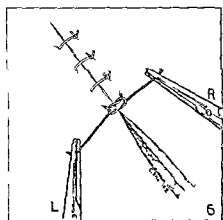


FIG. 329

No-touch technique—tying sutures and ligatures with forceps

No surgeon should operate on fractures unless he is a master of no touch technique including the tying of ligatures and sutures by two pairs of forceps. After sterilisation the threal or gut is never touched by hand.

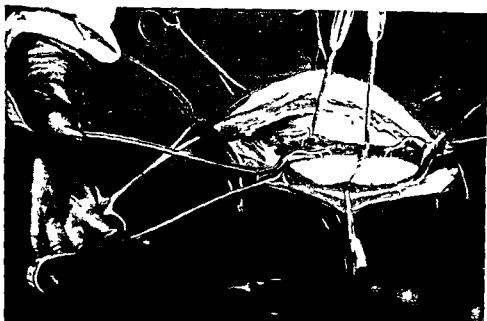


FIG 330



FIG 331

No-touch technique—field of operation

Open reduction and internal fixation with plate and screws. Note that the patient's skin is completely screened with side towels; the surgeon and assistants' hands are kept well away from the wound by long-handled forceps and retractors (Fig. 330); the plate is held with special forceps and the screws with special screw drivers so that neither are touched by hand (Fig. 331).

ligatures are necessary they are tied with two pairs of forceps, neither surgeon nor assistant ever touching the catgut by hand. Swabs of dry gauze used for mopping the wound are not touched by hand but only with forceps, the assistant is never without a pair of forceps in one hand for this purpose. Long retractors are used so that hands are well away from the wound. Needles threaded with forceps are held in needle holders and sutures are tied with forceps. Very occasionally it is necessary for the surgeon to palpate the deep tissues for example, to examine the density of a nerve bulb or determine the nature of a tumour.¹ He must first rinse his gloves in strong antiseptic and then in saline, it is an event—an unusual event—which occurs once in many scores of operations. Under no other circumstance must the hands of sister assistant or surgeon come in contact with the wound or with anything which will be put into the wound.

Internal fixation—If a fracture has been reduced and it is proposed to use internal fixation with screws or plates special precautions of asepsis have been recommended.² A small steriliser is kept on an instrument table with water on the boil throughout the operation. All drills screws plates or nails are left in this until required. Screw holders plate holders and screw drivers are put in the steriliser with their handles projecting over the edge before they are actually used in the wound. After use, these instruments are returned to the steriliser until required again. The end of the screw driver is plunged in the boiling water after the insertion of each screw. If all non absorbable foreign bodies and all instruments which come in contact with them, are reboiled in this way immediately before use the dangers of air borne infection are minimised.

Blood-borne infection—The aseptic technique which has been outlined aims at excluding infection from (a) dust and air (b) instruments and towels, (c) surgeon's and assistants' skin and (d) patient's skin. It is also possible for a clean wound to be infected by organisms carried through the patient's blood from a distant focus of infection. Although this is possible it is extremely rare. The incidence must be the same as the incidence of spontaneous infection of closed fractures and how many surgeons have seen more than two or three such cases in a lifetime.³ It is of course, wise to defer an elective operation if the patient is suffering from boils, tonsillitis or similar infections. But the possibility of blood borne infection can seldom be eluded as the explanation of post-operative infection. If there is an obvious distant source of infection the operation should not have been performed, and if there is no obvious distant focus the possibility is very remote indeed.

Lymph-borne infection—Although blood borne infection is rare, lymphatic infection is common if operations are performed in the region of pressure sores or unhealed wounds. It is not enough that the pressure sore is healed, bacteria remain in the lymphatics long after the skin is repaired. It is not enough that the operation is performed at a distance from the recently healed pressure sore, the risk is grave if there is any possibility of lymphatic communication. The operation should be deferred as long as possible, and certainly for not less than six or eight weeks.

¹ In draining an infected wound palpation with the finger is often wise—the most gentle touch is needed in exploring the walls of the cavity and ensuring that every unbroken track has been widely dilated. More over operations on the fingers often demand the use of fingers and hand in the wound.

² H. A. T. Fairbank. The Non touch Technique. *Brit Med Jour* 1911, II 394.

METHODS OF INTERNAL FIXATION

The fracture is exposed, callus is cleared from the fractured surfaces to restore the original serrations, and the fragments are locked by angulating them into position. Occasionally this is all that is necessary. If there is very firm natural stability of the fragments it may be possible to avoid the introduction of foreign bodies. Nothing is more unfortunate, however, than to perform an open reduction, decline the opportunity of ensuring perfect



FIG 332



FIG 333

Fixation by onlay bone graft

Recent fracture of the shafts of both forearm bones. Manipulative reduction is unsatisfactory (Fig 332). Remanipulation might improve the position, but in view of the unstable type of fracture and the likelihood of redisplacement, particularly of the ulna, internal fixation is better. This has been secured by onlay bone grafts with vitalium screws (Fig 333).

position by internal fixation, and three or four weeks later find that redisplacement has occurred. If there is doubt as to the stability of the fragments, it is better to use internal fixation. The methods which are available include, in their order of preference, (a) an autogenous onlay or inlay bone graft, (b) a single obliquely placed screw, (c) a plate and screws. Other methods which are not recommended are circumferential catgut, wire or metal bands, and intramedullary beef bone or ivory pegs. Whichever device of

internal fixation is used, it is to be regarded as a suture of bone and not as a splint for the bone, it supplements but does not take the place of external splints and plaster. Recently attempts have been made to fix fragments so securely that no external fixation is needed, non weight bearing exercises are practised from the beginning. This necessitates the insertion of much more foreign material, of heavier plates and more screws, and it carries a risk which in the present state of our knowledge of surgery and metallurgy is scarcely justified.



FIG 334



FIG 335

Fixation by inlay bone graft

Mal united fracture shaft of radius (Fig 334). There is 90° of rotation of the fragments, the antero posterior plane of the wrist coincides with the lateral plane of the elbow. After operative reduction an inlay bone graft was used for internal fixation (Fig 335). Correction of angulation and rotational displacement has restored radio ulnar movement.

Autogenous bone graft—A skilfully performed bone grafting operation provides perfect internal fixation. Although the fracture is relatively recent and may be in no danger of failing to unite, a bone graft has the great advantage of facilitating instead of hindering repair, and of being absorbed in the process. The technique described in the treatment of un-united fractures is followed (pp 38-45). For small bones like the radius and ulna, an onlay bone graft should be used with vitallium screws (Figs 332-333). In the case

of larger bones the surgeon may choose between onlay and inlay grafts vitallum screws often being necessary even with inlays (Fig 105)

Obliquely placed screw—The less foreign material introduced in the region of the fracture the better. Since the internal fixation is no more than a bone suture which supplements external fixation by preventing shearing and rotation movements oblique and spiral fractures can be dealt with very satisfactorily by means of a single screw transfixing the fragments (Figs 336 337). In the case of very long oblique fractures it is sometimes advisable to use more than one screw. The fracture is reduced and the fragments are



Fig 336



Fig 337

Fixation by obliquely placed screw

Five weeks old fracture of the shaft of the radius with inferior radio ulnar dislocation. This injury is very unstable. Redisplacement within the plaster has been prevented by one vitallum transfixion screw.

held with a Lowman's or Bendixen bone clamp. The correct axis of the screw must be carefully chosen as nearly as possible at right angles to the plane of fracture and through an equal thickness of bone on each side. The track is drilled. It is important that the drill should be exactly equal to the root diameter of the screw, if it is wider than this the screw threads will not fully engage in the bone. The exact length of screw required is estimated from the penetration of the drill. The screw is driven home snugly but not tightly. To prevent subsequent loosening the screw should be of non-corrosive non-electrolytic metal such as vitallum^{1 2}. It should be a self-tapping metal type of screw, not a pointed conical wood variety which engages less tightly at the point than at the head. Since the screw is completely buried and is non-toxic, it is unnecessary to remove it.

¹ Venable "Osteosynthesis in the Presence of Metals" *South Med Jour* 1934 XXXI 401
² Venable and Stuck "Fractures," *Jour Indiana State Med Assoc* 1934 XXXI, 342

Plate and screws—When the fracture line is horizontal one screw cannot transfix the fragments and if for any reason bone grafting is not employed a plate and not less than four screws must be used (Figs 338-339). The points to be observed in the operation are (1) absolute asepsis and no touch technique (2) accurate reduction of the fracture and the use of a good bone clamp to grip the fragments (3) a strong plate of non-corroding metal (4) self-tapping screws of the same metal as the plate to prevent electrolysis (5) drill of the same dimension as the root diameter of the screws (6) drill holes perfectly centred in the holes of the plate (7) at least one screw

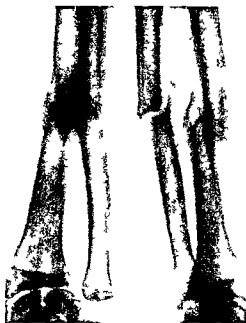


FIG 338



FIG 339

Fixed on by plate and screws

Mal united fractures of forearm bones with overriding of radius and inferior radio ulnar dislocation. The radius required open reduction and in view of the instability a stainless steel plate and screws was employed. This is less satisfactory than accurate bone grafting but more effective than an imperfect graft.

on each side of the fracture long enough to engage with the opposite cortex (8) screws firmly but not tightly driven home, (9) suture of periosteum but not of muscles and (10) complete external fixation by plaster casts.

Encircling suture with wire or band—It is tempting to immobilise a spiral or long oblique fracture with encircling wire or a Parham's band. Perfect fixation can be secured but the technique is unsafe. The pressure of the metal causes absorption of the underlying bone and refracture at this level has been reported many times. If the device is used it is advisable to remove the metal as soon as the fracture is united.

Encircling catgut suture—Even a strong catgut suture tied tightly round a bone can cause sufficient bone absorption to produce an almost spontaneous

fracture In one personal case after a leg shortening operation where fixation of the Z cut fragments had been secured by circular sutures of No. 3 catgut a fracture of the femur was sustained exactly at the level of one suture five months after operation while the patient was walking in a calliper splint with guarding leather

Intramedullary ivory peg—The insertion of an ivory peg into the medulla



FIG 340



FIG 341

Internal fixation with mass of beef bone intramedullary peg. This is not recommended. Ten years later (Fig 341) the peg is still incompletely absorbed.

of the fragments secures accurate apposition and guards to some degree against angulation but the peg blocks an important source of granulation tissue growth and delays repair. Moreover it is an inadequate bone suture for it does not prevent rotatory movement of the fragments. Small pegs are absorbed and disappear but large masses of ivory are incompletely absorbed and remain as an inert mass in the bone (Figs 340 341)

CHAPTER V

OPEN AND INFECTED FRACTURES AND WAR WOUNDS

For many years the surgery of wounds and open fractures has been dominated by a ritual of antiseptic therapy. The object of treatment was the destruction of bacteria by chemical agents. Coal tar derivatives, dyes, halogens, mercurial preparations, acridine compounds and other antiseptics were employed. First aid workers applied iodine and medicated dressings, casualty surgeons tried to sterilise the wound, after treatment included dressing with antiseptic lotions, cleansing with hydrogen peroxide, packing with bismuth iodoform paste and flooding with Carrel Dakin irrigations. Day after day and even week after week the antiseptic attack was continued.

The limitations of antiseptic therapy—It is not difficult to prove that such treatment was both unscientific and unsuccessful.

1 A general protoplasmic poison such as phenol is fatal to all non sporing micro organisms but less destructive antiseptics may be almost specific in their action. Staphylococci are intensely susceptible to violet dyes whereas streptococci are less susceptible and gram negative bacteria are highly resistant. Haemolytic streptococci are inhibited by the acridine compounds but staphylococci are not. *B. pyocyaneus* flourishes in the presence of most antiseptics but is destroyed by 1 per cent acetic acid to which other bacteria are resistant. These selective actions may be of practical value when the organism has been identified but the routine use of one antiseptic for all infections and the continued use of antiseptics without regard to bacteriological findings is obviously irrational.

2 An antiseptic may be capable of destroying micro organisms while they lie on the surface of a wound but within a few hours of injury they no longer lie on the surface. The tissues have then been invaded and enormous numbers of bacteria are multiplying in deep cellular layers, in fascial planes and intermuscular spaces far beyond the reach of surface applications. The limit of tissue penetration for most antiseptics is a fraction of a millimetre¹ and even if Carrel Dakin irrigating tubes are buried in the recesses of a wound cavity they are still on the surface of the wound, remote from the deep layers in which the organisms lie.

3 Even the control of surface infections is lost within twenty four to forty eight hours because the continued application of any noxious agent which is not wholly lethal allows only the more resistant bacteria to survive. Succeding generations acquire an increasing resistance until they lose all susceptibility and become habituated.

4 Not only are antiseptics limited in their power to destroy bacteria and certainly incapable of destroying bacteria they cannot reach but they

¹ L. P. Garrod. Action of Antiseptics on Wounds. *Lancet* 1940 1 845

are toxic to the cells of the invaded tissue as well as to the bacteria which are invading the tissue. The more potent an antiseptic in destroying bacteria the more certain is its destruction of leucocytes. The normal defences against infection depend upon leucocytes. Furthermore destruction of leucocytes and other living cells liberates tryptic ferments reduces the antitryptic quality of blood and blood fluids and reduces the inhibitory power of these fluids over the growth of sero saprophytes which include nearly all the organisms of primary wound infection.

Antiseptics are harmless when applied to the resistant surface of the skin they are of value in pre operative skin preparation when applied before the infliction of a wound and of possible value in first aid treatment when applied shortly after the infliction of a wound but when applied repeatedly day after day and week after week to the delicate cellular surfaces of injured tissues they are both useless and harmful.

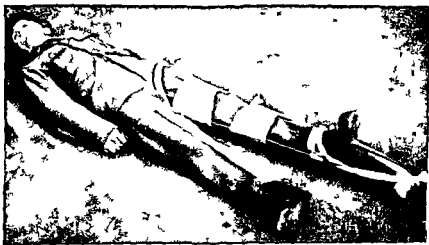


FIG 342

The Thomas splint is the ideal first aid splint for any lower limb fracture. It is applied before removing the boot cutting the trousers or dressing the wound.

Principles of the treatment of wounds—The field of antiseptic treatment is prophylaxis. After the first few hours treatment must be concentrated not on destruction of the invading organism but on promotion of the normal defences. Behold a sower went forth to sow. And it came to pass as he sowed some fell on stony ground where it had not much earth and when the sun was up because it had no root it withered away. And other fell on good ground and sprang up and increased some thirty some sixty and some an hundred. A wound with necrotic debris fragments of clothing and other foreign bodies in its depth and with crushed avascular devitalized tissue in its wall is good ground in which the seed of bacteria will spring up and increase. A wound protected from further soft tissue injury by immobility from which foreign matter and dead tissue has been excised with vascular pulsating walls of living cells free from the necrosis of injurious chemicals is stony ground in which the seed cannot take root.

First-aid treatment—The primary object of first aid treatment must be

the protection of soft tissues from further injury and devitalisation by the immediate application of splints. In the lower limb a Thomas splint is applied before the boot is removed or the trousers cut¹² and before the wound is exposed (Fig 342). A clove hitch round the ankle, a metal skewer through the boot or a Millbank clip on the waist of the boot (Fig 343) is used to pull down the foot towards the end of the splint. In the upper limb Cramer wire metal gutters or improvised splints are employed. Dressing of the wound and the application of antiseptics are of secondary importance.

Emergency treatment—The general resistance of the patient must be increased by the treatment of primary shock, wound shock and hæmorrhage. The local resistance of the injured tissues must be promoted by early excision of the wound. This emergency operation can seldom achieve sterilisation of the wound. Its object is not simply to remove infected tissues but to prevent

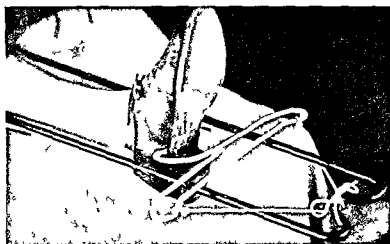


FIG 343

The Millbank clip by which traction can be applied through the patient's shoe or boot.²

the survival of bacteria which remain by restoring a free blood supply to every wall of the wound, removing foreign bodies and necrotic debris, excising dead and dying tissue, controlling hæmorrhage, dividing deep fascia to permit swelling of muscles without strangulation, and in late cases by establishing free drainage in order to prevent tension and impairment of circulation.

After treatment—The purpose of after treatment is again to promote both general and local resistance. general resistance is increased by anti-toxin therapy and chemotherapy, local resistance is increased by continued

¹² In the war of 1914-18 the introduction of the Thomas splint to the front line and its application even before the dressing of wounds reduced the mortality of gunshot fractures of the femur from 80 to 70 per cent. Ambrose Lockwood. *Some Experiences in the Last War*. *Brit Med Jour* 1919 1, 356. By this simple and ingenious device the foot can be pulled down towards the lower end of a Thomas splint. It springs on the margin of the sole just below the instep, is much easier to apply than a skewer through the boot, can easily be applied by sense of touch in the black-out, and automatically keeps the foot up at the right angle and prevents rotatory movement of the limb. The original clip was devised by Dr. Pictor (*Ind Med Jour* 1939 1, 1914) and is copied by Lt. Col. Monroe (*Brit Med Jour* 1910 1, 1).

immobility of injured bones and soft tissues elevating the limb to prevent oedema and avoiding the tissue necrosis of repeated dressing with injurious antiseptic chemicals¹.

PRIMARY SHOCK, WOUND SHOCK AND HÆMORRHAGE

Primary shock is a condition of collapse which may occur after any injury. It is similar to fainting and responds rapidly to treatment by recumbency, warmth, relief of pain and the administration of stimulants. More severe injuries which are accompanied by extravasation of fluid into tissue spaces or hæmorrhage and fluid loss from open wounds cause secondary or wound shock which develops insidiously after some hours and may prove fatal⁶.

Physiological basis of wound shock—Reduction in blood volume due to fluid loss causes general vasoconstriction which usually succeeds in main-

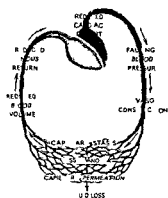


FIG 344

The death cycle of wound shock

taining a normal blood pressure if the loss of volume is less than 2 pints. If the loss is greater the blood pressure falls and the combined effect of intense vasoconstriction and falling blood pressure is to diminish the tissue circulation. If this is not restored rapidly oxygen lack causes permanent tissue damage with increased capillary permeability and in escape of plasma into tissue spaces^{7,8}. The permeation of colloids increases the osmotic pressure in extravascular spaces until it is greater than that within the blood vessels and fluid loss may then continue even despite replacement of the blood by transfusion. The death cycle of shock is represented in Fig 344. Treatment must begin before this vicious circle is established.

Three degrees of wound shock may be differentiated clinically —

	Group I	Group II	Group III
Clinical condition	Good	Serious	Dangerous
Pulse (per minute)	90-110	100-140	120-160
Systolic pressure	Over 90 mm	70-90 mm	60 mm or less
Blood volume	Over 75 per cent	65-75 per cent	50-65 per cent

The normal blood volume is 9 pints and whereas a loss of about 2 pints which reduces the volume to 75 per cent of normal may cause only slight symptomatic disturbance, a further loss of 2 pints which reduces the volume to 50 per cent gives rise to abnormal capillary permeation and a condition so serious that recovery is exceptional. The restoration of blood volume is therefore a matter of urgency and the 1 pint transfusions of the past were inadequate. In mild cases of hæmorrhage and wound shock

¹ Winnett Orr "Osteomyelitis and Compound Fractures" London, 1909

² Trueta and Barnes "Immobilisation in the Treatment of Infected Wounds" *Brit Med Jour* 1940 II 46

³ Trueta "Treatment of War Wounds and Fractures" London 1939

⁴ D. W. Jolly "Field Surgery in Total War" London 1940

⁵ d. Harcourt Folch and Oriol "Closed Plaster Treatment in Spanish War" *Brit Med Jour* 1940 I 812

⁶ "The Treatment of Wound Shock" Medical Research Council Memorandum 1940 II 31 Stationery Office

⁷ R. J. McDowell "The Circulation in Relation to Shock" *Brit Med Jour* 1940 I 919

⁸ K. W. Raven "Syndrome of Traumatic Shock" *Post grad Med Jour* April 1940 112

at least 2 pints of blood are required and in severe cases 3 or 4 pints are necessary. Recent work indicates that transfusion of solutions of dried blood plasma or serum is even more effective than transfusion of whole blood¹³. There is no lack of red blood cells—on the contrary the blood is already concentrated by the escape of plasma. The important loss is that of plasma protein and not until this is restored and the osmotic equilibrium regained is continued fluid loss prevented.

Clinical recognition of wound shock—Profound shock is easily recognised by the rapid thready pulse weakness coldness pallor cyanosis of lips ears and nails cold sweating dry tongue vomiting and intense thirst. Recognition is more difficult in the early stages at the time when treatment must be instituted. It should be suspected in every case of severe injury where the skin is pale and cold and the pulse rate is over 100. A ten or fifteen minute pulse chart should be kept and differentiation of the degrees of shock based on the rising pulse rate and general clinical signs rather than on the falling systolic blood pressure. Even in severe shock the blood pressure may be sustained temporarily by intense vasoconstriction only to fall suddenly and rapidly if the shock is slightly intensified by anaesthesia or a further small loss of blood.

A more accurate early diagnosis can be made by blood examination. An increase in the cells to plasma ratio or a rising haemoglobin percentage indicates haemo concentration due to loss of plasma through the capillary walls¹⁴ and is the earliest sign of wound shock. It differentiates shock from uncomplicated haemorrhage where there is haemo dilution and it is an index of the severity of the shock. Haemo concentration of 120 per cent is ominous and of 140 per cent grave. In the terminal stages shortly before death it may rise to 160 or 180 per cent.

Treatment of Wound Shock

More lives may be saved by first aid in the resuscitation ward than by skilled surgery in the operating theatre. Secondary shock is usually preventable and although the emergency operation of wound excision is urgent the treatment of shock must take precedence during the first few hours after injury.

First-aid treatment includes (1) the arrest of haemorrhage by firm pressure bandaging over the wound and sometimes but not often by a tourniquet above the wound (2) immediate splinting of the limb to prevent pain and local plasma loss due to movement of a fractured bone (3) protection from cold by blankets under as well as over the patient (4) raising the foot end of the stretcher (5) giving water or tea freely and repeatedly (except in the case of abdominal wounds) with half a teaspoonful of salt to the pint of fluid and (6) relieving pain and restlessness by morphine labelling the patient with the dose and the time that it was administered. The arrest of haemorrhage is of great importance but local pressure should usually be relied upon and first aid workers must be warned of the dangers of tourniquets. Inadequately applied tourniquets increase the haemorrhage and

¹³ Knott and Kerner "Storage of Transfusion Plasma" *Lancet*, 1937, ii 1069

¹⁴ R. Edward, J. Kay and T. B. Dale "Preparation and Use of Dried Plasma" *Brit. Med. J.*, 1940, i 3

Levinson, Newell and Necheles "Human Serum as a Blood Substitute in the Treatment of Haemorrhage and Shock" *Jour. Amer. Med. Assoc.*, 1940, cxlv 455

¹⁵ J. C. Makin "Shock—its Causes and Treatment" *Canad. Med. Assoc. Jour.*, 1940, xli 101

¹⁶ H. Moon "Early Recognition of Shock I Retention from a Haemorrhage" *Ann. of Surg.*, 1937, cx 960

overlooked tourniquets may cause gangrene (p 129) More limbs and lives are lost by the improper use of the tourniquet than are saved by its proper use ' (Douglas Jolly) ¹

Treatment in the resuscitation ward—The ward must be well heated and as quiet and sheltered as possible Wounded men are exhausted from pain sleeplessness fear and prolonged exertion before wounding, they require complete rest Wet and dirty clothes are cut off warm clothes are put on, and the bed is heated by hot bottles hot bricks radiant heat baths or electric blankets The foot of the bed is raised by blocks about 12 in high

Relief of pain and restlessness—If pain is severe and no hypnotic has already been given $\frac{1}{2}$ or $\frac{1}{4}$ gr of a morphine salt is administered subcutaneously When the circulation is feeble the intravenous injection of $\frac{1}{2}$ or $\frac{1}{4}$ gr in 1 c c of sterile water has an immediate effect but it must be given slowly, the injection occupying at least one minute Morphine is often given too sparingly to be of real value but since it may cause depression of the respiratory and cardiovascular systems a full dose should not be followed by a second dose in less than four hours Alternatively, the less depressing preparations dihydrid ($\frac{1}{10}$ to $\frac{1}{15}$ gr) or omnopon ($\frac{1}{2}$ to $\frac{3}{4}$ gr) may be used

Correction of dehydration—Every shocked patient who is not unconscious or suffering from abdominal wounds must be urged to drink repeatedly and copiously Warm sweetened tea or coffee is better than water and it should contain half a teaspoonful of salt to the pint If the patient is unconscious or there is persistent vomiting rectal salines should be given or intravenous glucose salines with 10 units of insulin to the pint ²

Administration of oxygen ³—Oxygen in high concentration helps to relieve tissue anoxia and it should be given by an inhalation apparatus to all patients with cyanosis of the lips ears or nails especially if the respiratory oxygen intake is reduced by pulmonary oedema or chest injury Injuries due to explosion in enclosed places may be complicated by carbon monoxide poisoning and require the inhalation of oxygen with 5 per cent carbon dioxide to promote deep breathing

Restoration of blood volume—The earlier the blood volume is restored the better the prognosis From 2 to 4 pints of whole blood ⁴ or human plasma are required according to the severity of the shock ⁵ The transfusion must be given with the least possible delay and it may be continued as a drip transfusion throughout the operation of wound excision ⁶

Unclotted blood transfusion—late late of the react —For the first few days after massive blood transfusion the possibility of hæmolytic due to mismatching of blood must be borne in mind The symptoms are secondary collapse with a cold clammy skin dyspnoea and vomiting followed by transient jaundice increasing oliguria and finally anuria due to blocking of the renal tubules with acid hæmatin from the foreign hæmoglobin The urine must be alkalised a mixture of potassium citrate (gr xxx) sodium bicarbonate (gr xxx) syrup (mxxx) water to 1½ or is given orally four or five times in twenty-four hours If anuria persists for forty-eight hours saline with 3 per cent potassium citrate and 3 per cent sodium bicarbonate should be given by intravenous drip the saline transfusion is to be continued as soon as possible because there is danger of pulmonary oedema and cerebral fulminant

Treatment of the wound—Absorption of the toxic products of bacterial infection lowers the patient's vitality and aggravates shock, so that surgical

Jolly Field Surgery in Total War London 1940 ¹Eller Surg Gyn Obst 1916, all 11 21
 M D Leitch and Richardson Oxygen Therapy and its Variants (Cambridge Med Jour 1914 xlvii 111)
 * Edwards & T Davie Preserved Blood—Report from Mersey-side Blood Bank Brit Med J 1940 ii, 3
 The technique of blood and plasma transfusion is fully described in the Medical Research Council's War Memorandum No 1 The Treatment of Wound Shock 1940 H M Stationery Office
 * H H Riddell Blood Transfusion London, 1939



FIG 345

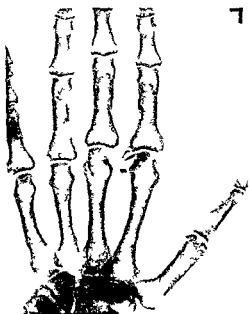


FIG 346



FIG 347

Typical foreign bodies of war wounds

FIG 345.—A piece of tubular glass in the palm. FIG 346.—Fragments of wood in the hand of a bombler. The shadows are dense because the fragments are pieces of plaster lith from a roof. FIG 347.—Typical peppered wound with many metallic fragments tattooed into the soft tissues.



FIG. 348



FIG. 349

FIG. 350

Typical foreign bodies of war wounds

FIG. 348—Cannon shell wound with shattering of bone, many metallic fragments, and bubbles of air blown into the tissues (not gas gangrene). FIG. 349—Missile bomb wound; the foreign body (shown in the inset) is the cap of the bomb. FIG. 350—Incendiary bullet wound, after incision of the wound and entry of a combustible phosphorus incendiary. Symptoms of phosphorus poisoning have been recorded in such wounds. (A. J. Blount, *Brit. Med. J.*, 1916, ii, 664; and J. W. Little, *Brit. Med. J.*, 1916, i, 6.)

treatment of the wound must not be postponed too long One of three surgical procedures is indicated —

- (1) If the operation is performed within twenty four hours of injury—excision of the wound and light packing with gauze (or in special circumstances suture of the skin but not of the deep layers)
- (2) If the operation is performed later than twenty four hours after injury—free incision of infected tissues and drainage by light packing with gauze
- (3) If the main blood vessels are destroyed and gangrene is imminent or there is massive gas gangrene—amputation

TREATMENT BEGUN DURING THE FIRST TWENTY-FOUR HOURS AFTER INJURY—EXCISION OF THE WOUND

The technique of operation is similar in wounds of soft tissues wounds involving bones (open fractures) and wounds involving joints (open dislocations and penetrating joint wounds) The principles of treatment are the same whether the injury is the result of domestic accident motor car collision bullet wound bomb or shell explosion or falling masonry In normal civilian life it is customary to distinguish fractures compound from within where the skin is simply punctured by a sharp bone fragment from fractures compound from without where the skin and muscles are crushed and the wound is probably contaminated In war time such a distinction is dangerous Bomb fragments often cause small punctured skin wounds which nevertheless are associated with severe internal injury and gross contamination

Preliminary radiographic examination—Accurate pre operative localisation of foreign bodies is of assistance in planning the operation of wound excision (Figs 345 350) Whenever possible wounds should be radiographed before excision particularly if the probable cause of injury was flying glass or bomb fragments and there is a wound of entry but no wound of exit Repeated radiographic examination may be necessary

A civilian was injured by bomb explosion The wound of entry was in the upper thigh below the greater trochanter but there was no wound of exit Radiographs of the hip joint and pelvis showed no evidence of foreign body Further radiographs of the whole length of the thigh were also negative A third series showed a bomb fragment in the left upper quadrant of the abdomen Exploration revealed a punctured wound of the descending colon which was successfully sutured Without full pre operative X ray investigation the surgeon would have been faced with an almost impossible task in his effort to follow the track of the wound

Anæsthesia—Gas and oxygen with ether is probably the anæsthetic of choice but since the reduced oxygenation of tissues which is characteristic of wound shock is aggravated by cyanosis and asphyxia gas should be given only by an expert anæsthetist In other circumstances open ether is to be preferred Chloroform is dangerous and spinal anæsthesia very dangerous Many patients have died on the operating table owing to the use of spinal anæsthesia for operations on war wounds of the thigh and other injuries associated with profound shock

Tourniquet—No tourniquet is to be used because with a bloodless field it is difficult to distinguish living vascular tissue which need not be excised from dead avascular tissue which must be excised. Moreover the blood supply of the wound must not be reduced even for the thirty or forty minutes of operation. Defence against infection depends almost entirely on the blood supply. The tissues are already devitalised and if they are completely deprived of blood for half an hour or more bacterial growth is encouraged and infection may be established.

Excision of Soft Tissue Wounds and Compound Fractures

Cleaning the skin—Clothes bandages and first aid dressings are removed the wound is covered with sterile gauze and the skin is cleaned over a wide area with ether spirit and an antiseptic such as C.T.A.B. or iodine. The surgeon then changes his gloves and screens the skin with sterile towels to within 1 in. of the wound margin. Skin seldom requires wide excision it is usually sufficient to remove a 1 or 2 mm. strip.

Excision of fascia and muscles—The skin wound must be enlarged in the long axis of the limb in order to expose deep tissues adequately and avoid the necessity for heavy retraction and burrowing in undermined pockets.¹ Incisions on each side of the limb may be needed for injuries due to explosives where there is a small wound of entry on one side and a large wound of exit on the other. Working systematically from superficial to deep layers the wound is searchingly explored and bruised tags of fascia and crushed muscle are cleanly excised with a sharp scalpel or scissors. Alteration in the appearance and colour of muscle, loss of contractility on mechanical stimulation or failure to bleed demands wide excision of this most vulnerable tissue particularly when gas gangrene is suspected. Intermuscular spaces are opened and blood clot is evacuated. Deep fascia is freely divided in the long axis of the limb in order to permit the subsequent swelling of muscles without stranguation. Care must be taken to avoid injury to main blood vessels and nerves.

Excision of foreign bodies and bone—Fragments of bomb-crang shell bullet clothing leather wood grass mud or other foreign bodies must be removed. Bone fragments completely denuded of soft tissue attachments are carefully lifted out but no bone with adherent soft tissue is to be removed. No matter how flimsy the attachment and no matter how precarious the blood supply bone must not be removed if it is not completely loose. There must be no question of dissecting the bone, or roughly twisting it from the wound. Excision of whole segments of the shaft of a long bone is particularly serious owing to the danger of non union and prolonged incapacity (p. 34). The bone must not be scrubbed.² No strong antiseptics are introduced the wound is not irrigated and it is not curetted.

Burying foreign bodies in the wound—Since one of the main objects of operation is the removal of foreign bodies it is difficult to find justification for the burial of other foreign bodies such as wire screws or plates. Even sterile catgut is an irritant which causes sero fibrinous exudation and increases the danger of infection. The less catgut buried in the wound the greater

W. H. Ogle. Surgery of Infected Wounds. *Lancet* 1910. 1. 935-7. 975-7.

¹ It has even been recommended that the whole wound should be scrubbed with soap and water! 4 of a practice is a full hit of the antiseptic era when the object of treatment was the destruction of bacteria at all costs even at the cost of destroying living tissue. It is the stability of the wall of the wound which is of vital significance and any treatment so dangerous as the scrubbing of tissues is obviously wrong.



FIG 3.1

Compound fracture of the shafts of both bones of the forearm treated by prompt excision of the wound

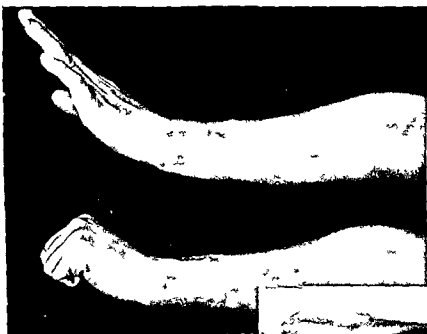
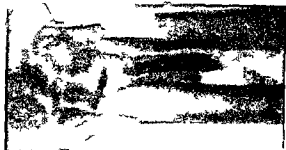
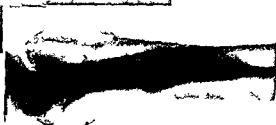


FIG 3.2

Early operation prevented infection. There is normal finger movement and practically no disability. The fractures are united in good position.



Excision of soft tissue wound and compound fracture

the likelihood of first intention healing. Haemostasis can usually be achieved by applying artery forceps to every bleeding point and twisting them off at the conclusion of the operation. Only large vessels need ligation with thread or fine catgut.

Suture of nerves and tendons—Severed nerves and tendons should not be sutured except in the case of cleanly incised wounds due to flying glass which will heal by first intention. Only when the surgeon is quite sure that the wound will heal without infection is he at liberty to suture divided nerves. Even exploration with the object of deciding whether or not a nerve is divided is seldom advisable in contaminated wounds. If a wound becomes infected no advantage has been gained by the suture of nerves or tendons and it may well be that if suture material had not been buried infection



FIG 303

Compound fracture of both leg bones treated by excision on vaseline gauze pack drainage and closed plaster. There was severe infection and complete immobilisation was necessary for ten months (for radiographs see Figs 307)

would have been avoided. It is better to concentrate on thorough excision and to perform secondary suture after the wound is healed (Figs 303-304).

Suture of muscles and deep fascia—In no circumstances is it permissible to suture fascia, muscles, periosteum or other deep layers of the wound. Not only is catgut an irritant foreign body which promotes infection but swelling and expansion of tissues must be permitted without risk of tension and impairment of the circulation. It is particularly important therefore to avoid the suture of fascia. Free division of this membrane is an important step of the operation of wound excision; certainly it must not be sutured.

Prophylactic chemotherapy—When wound excision is complete a measured quantity of powdered sulphonamide is scattered evenly throughout the cavity or blown in with an insufflator. From 5 to 1 gm is used

See also the use of sulphur in the treatment of infected wounds and the use of antibiotics in the treatment of infected wounds.

according to the size of the wound. Prophylactic chemotherapy must not, however, take the place of wound excision. The protection of chemotherapy is totally inadequate as compared with the protection of surgical excision. Moreover, there can be no support for the view that when chemotherapy is available the operation of wound excision is no longer urgent and can be performed at leisure. This view is unsupported by clinical experience and untenable on theoretical grounds. Experimental evidence shows that the action of sulphonamides is inhibited by dead muscle¹ and inhibited by pus fluid.² Chemotherapy supplements but does not take the place of wound excision. It is an added protection especially valuable in controlling the growth of hemolytic streptococci and *Cl. welchii* and most effective when given before infection is established. As an alternative to local wound treatment the



FIG 304

Same as Fig 303. The tibialis anterior and extensor hallucis longus tendons had been severed and were repaired after eighteen months. The patient now pursues all normal activities and recreations.

drug may be given by mouth—2 gm. as soon as possible after injury, followed by a $\frac{1}{2}$ gm. tablet four hourly for a period of from seven to fourteen days.³

Suture of the skin—In sucking wounds of the chest and penetrating wounds of the brain immediate skin suture is imperative, in wounds of joints skin suture is often advisable. In other wounds skin suture is never imperative and seldom advisable. If the wound does not become infected the gain resulting from suture of the skin is slight acceleration of healing and perhaps a neater scar. If the wound does become infected the loss resulting from suture of the skin may be spreading infection, gangrene, amputation or death. No matter how skilfully an excision has been performed the surgeon can never be certain whether the wound will become infected or not. Is he justified in gambling when the stakes are a neat scar against an

¹ M. C. Nelson and N. K. Jensen, *Surp. Gyn. Obst.* 194, lxxxv, 31.

² A. Fleming, *Proc. Roy. Soc. Med.* 1941, xxxviii, 1.

³ A. Fleming and G. Janes, Dosing of Sulphonamide in Wound Infections, *Lancet* March 1940, 497.



FIG 355



FIG 356

Closed plaster technique for excised wounds

After excision and removal of foreign bodies and tags of devitalised tissue (some of which are seen on the drape) the wound is lightly packed with vaseline gauze (Fig 355). The skin is protected with vaseline gauze (prepared in a bandage roll) and plaster is applied (Fig 356).

amputated limb? The answer cannot be in doubt. In the cleanly incised and relatively uncontaminated wounds of normal civilian life immediate suture of the skin after wound excision may be safe. In penetrating crushing wounds and in the wounds of war immediate suture of the skin cannot be safe. Suture of the skin is very seldom justified in wounds of the fingers there is so little margin for swelling that tension and therefore infection is almost certain. Moreover excellent apposition of the skin margins can be secured by bandaging alone. Suture of the skin is never justified in wounds of the foot. I have never yet seen first intention healing of a severe penetrating wound of the foot. Suture of the skin has no real advantage in wounds and compound fractures of the leg thigh and upper limb. If it seems certain that the wound will heal without infection cover it with gauze and let it heal. If it is not certain that the wound will heal without infection gently separate the walls with a light gauze pack and let it heal. In neither case take the unnecessary risk of suturing the skin. At all costs do not half suture the skin. Do not lay in strips of rubber which admit infection even more certainly than they drain it.

Application of plaster.—A long line of menders of the maimed in British medicine—Hunter, Hilton, Hugh Owen Thomas and others—taught that inflamed tissue is capable of dealing with its enemies unaided if kept at rest. The merits of the closed plaster treatment for wounds already infected was proved beyond doubt by Winnett Orr. The merits of the closed plaster treatment for excised wounds in which it is hoped to avoid infection has been proved beyond doubt by Trueta. If the blood supply of the limb has not been jeopardised if the wound has been adequately excised and no liver has been sutured—neither muscle fascia nor skin—the ideal treatment whether the injury is a wound of soft tissues or a compound fracture is the application of a plaster cast which ensures complete rest (Figs. 355-356).

The probable explanation of the striking success of this treatment is indicated by recent experimental work.¹ Capillary endothelium of blood vessels is resistant to bacteria. Infection reaches the blood stream by the lymphatic circulation and any factor which diminishes the flow of lymph assists in localising the infection. If a limb is immobilised the flow of lymph almost ceases. It is muscle contraction and particularly joint movement which squeezes infection into the lymphatic meshwork. The joints above and below the wound must therefore be immobilised. No window should be cut in the plaster. There is no need to dress the wound. Indeed one of the great merits of enclosure of an excised wound in plaster is that it cannot be dressed. True capillary cells tear readily and wait with pouting mouths for further organisms. A dressing with the gentlest hands is then like the trappings of elephants' feet.²

Elevation of the limb.—As soon as the patient is back in bed the limb must be elevated. For at least ten days after operation it is kept on a plane higher than the trunk and never allowed to be dependent even for short intervals. Gravitational oedema increases tension within the plaster. It delays healing of the wound and it promotes stiffness of the joints. On the other hand joints which are susceptible to stiffness even joints of the fingers retain their mobility despite immobilisation in plaster for two or three weeks provided

¹ J. M. B. and J. Trueta, *Effect of Bacteria and Toxins from the Tissue*, *Lancet* 1941, 1, 61.
² *Lancet* 1941, Nov. 1st, p. 641.

that constant elevation of the limb is maintained so rigorously that the tissues are never once soaked in the sero fibrinous exudate of œdema

Excision of Bullet Wounds, Peppered Wounds and Shrapnel Wounds

There are three exceptions to the rule that the whole wound track must be excised and all foreign bodies removed. In wounds inflicted by hard



FIG 35

Lead shot wound sustained during clay pigeon shooting practice. Excision of all foreign bodies is obviously impossible.

bullets of high velocity it is often sufficient to excise the small wound of entry and the equally small wound of exit because the track is cleanly drilled the destruction of soft tissues is minimal and foreign bodies are seldom left in the wound. An Air Force pilot was shot in the palm of the hand and the bullet having travelled almost the whole length of the limb lay beneath the triceps muscle in the upper arm. Not one muscle tendon artery nerve or bone was severed. The bullet was removed and the wound in the palm excised but the track in the forearm and upper arm was not exposed. Healing was by first intention and despite deep hæmorrhage in the forearm and œdema of the hand due to median neuritis graduated finger exercise prevented stiffness or contracture and recovery was complete within eight weeks. In a second group of cases total excision is not indicated because minute foreign bodies are tattooed over a wide area (Fig 357). This peppering by tiny splinters causes minimal devitalisation of tissues and rapid

healing usually occurs despite the foreign bodies. In a third group larger metallic fragments are scattered so diffusely over the limb that nearly every muscle is involved and it would be an utter impossibility to excise every wound (Figs 358-359). Any of these fragments may cause devitalisation of tissues and therefore infection but the problem is to be met by incision and drainage rather than by excision.

Excision of Joint Wounds

The mobilisation of acutely inflamed joints which was advocated in the



Figs 3-8-3-9

Multiple "shrapnel" wounds

Over fifty metallic fragments are scattered throughout the thigh and leg. The minute fragments are unimportant, larger fragments cause devitalisation and therefore infection but they must necessarily be dealt with by incision and drainage, complete excision is impossible.



FIG. 360

Compound fracture of patella with wide opening of knee joint. The wound was excised. Only two catgut sutures were buried.

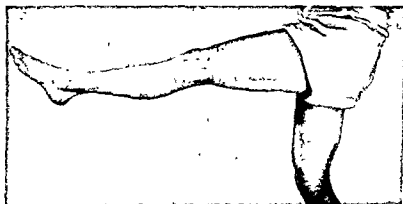


FIG. 361



FIG. 362



FIG. 363

Excision of joint wounds

Same case as Fig. 360. There has been no infection. Knee movement is satisfactory, there is normal power in the quadriceps and the fracture is firmly united.

last war by Willems has now been abandoned. It is as reasonable to attempt to cure a fever patient by kicking him out of bed as to benefit joint disease by wriggling at the articulation (Hugh Owen Thomas). If the principle of continued and uninterrupted rest is observed joints suffering grave injury and contamination are far less prone to disastrous sequelæ than was hitherto believed. This has been confirmed by recent experience with civilian casualties in bombed cities. Among the most gratifying of cases have been penetrating wounds of joints and particularly of the knee joint. These wounds are much less likely to become infected than wounds of muscle and cellular tissue. In view of this relative immunity there is justification for primary suture of joint wounds which have been excised within the first six or eight hours (Figs 360-363). A few interrupted sutures are inserted in the capsule and the skin is sutured. The limb should not be enclosed in plaster. The closed plaster technique is perfectly safe when wounds are widely laid open and drained but it is not safe when the wound is sutured. It is true that constitutional signs, the temperature chart and the pulse chart are the most reliable evidence of infection. But it should also be possible to inspect the region of the wound. It is better to use a Thomas splint or plaster slab until the danger period is passed. The second reason why sutured wounds should not be enclosed in plaster is that first intention healing is promoted by dryness of the skin and exposure to the air and it is hindered by the moisture of serous discharges and a sodden dressing. Maceration of skin round the sutures promotes secondary infection by skin organisms. If the wound is dried by exposure to the air this secondary infection is prevented and first intention healing is secured in a far higher proportion of cases. A clear distinction must therefore be drawn. Wounds which are not sutured may be enclosed in plaster with safety. Wounds which are sutured should not be enclosed in plaster—they should be freely exposed to the air.

If more than six or eight hours have elapsed between wounding and operation there is danger of infection of extracapsular cellular tissues but still a likelihood that the joint itself will remain free from infection. It is advisable therefore to excise the wound and suture the capsule but to leave the skin unsutured. A light gauze packing is inserted, the joint is immobilised and the limb is kept under careful observation. The alternative measure when there is doubt is to excise the wound, pack the joint and wound with gauze and prepare for secondary suture after ten days or a fortnight if no infection develops. Knee joints which have been widely packed open with vaseline gauze for two or even three weeks have regained normal movement and almost normal function after secondary suture. In other cases where a low grade infection has made it advisable to continue the gauze pack, closed plaster technique and secondary suture has not been performed, surprisingly good movement and function has sometimes been regained (Figs 368-370).

If more than twenty-four hours have elapsed since injury the opportunity for wound excision has passed. There must be no question now as to the correct line of treatment. Contaminated tissues are freely incised but not excised, the joint is drained, no layer is sutured, neither capsule nor skin, drainage of the wound and the joint is secured by packing with gauze, the limb is immobilised in plaster (Figs 364-367).



FIG. 364



FIG. 365

Closed plaster technique for infected joint wounds

Penetrating wound of the knee joint complicated by septic arthritis and treated by free incision into the front of the joint and suprapatellar pouch the posterior medial and the postero lateral compartments (Fig. 364). The wounds are lightly packed open with vaseline gauze and a plaster spica is applied (Fig. 365).



FIG 366
Five weeks after operation



FIG 367
Eleven weeks after operation
Closed plaster technique for infected joint wounds

After five weeks when the plaster is changed the wound looks unpleasant (Fig. 366) but the patient's general condition, temperature and pulse chart show that infection is controlled. Six weeks later the wound is almost healed (Fig. 367). Immobilisation in a plaster cast was continued until the joint was so rigid and locked



FIG 368



FIG 369



FIG 370

Excision of penetrating bullet wound of joint

Bullet wound penetrating the knee joint with destruction of femoral condyle and tibial tuberosity. The foreign body was removed and the joint lightly packed with gauze. No primary or secondary suture was performed. Nevertheless 100% of knee movement was regained with almost normal power and stability and full duty was resumed within eight months with an A1 category.

Excision of Skull Wounds

Wounds of the scalp, especially war wounds, are potentially serious because the wound is often more extensive than first inspection would suggest and if there is an associated injury to underlying bone and brain there is a strong predisposition to infection. Whenever possible these cases should be treated by experienced neuro surgeons in fully equipped theatres. Early excision of the wound is of vital importance particularly when there is evidence of underlying craniocerebral injury. Cruciate enlargement of the wound is inadequate; there must be wide exposure by means of large scalp flaps. Blood clot and extruded brain matter are removed by means of a sucker. Fragmented or devitalised bone is freely removed. Unlike compound fractures of the limbs where wide excision of bone has unfortunate sequelæ defects in the skull due to radical treatment can be repaired with relative ease. The dangerous sequel is not non union but infection. There must be no hesitation therefore, in removing bone which is contaminated or devitalised. The dura is opened. Damaged brain tissue, clot and bone fragments are sucked out of the cavity. Hæmostasis is secured by clips or diathermy coagulation. The dura is then sutured. The exposed area must be covered by sound scalp which has been carefully sutured. If scalp tissue has been destroyed in the region of the wound, flaps should be swung into position even at the cost of denuding other areas where periosteum and bone are uninjured; these areas heal by granulation and can be skin grafted at a later date.

Excision of Chest Wounds

A penetrating wound of the chest is a serious emergency. Two types of wound are to be differentiated: (1) valvular wounds which allow entry of air into the pleural cavity during inspiration but prevent its exit during expiration, thus causing a tension pneumothorax. (2) sucking wounds which allow free entry of air during inspiration, and free exit during expiration, thus causing an open pneumothorax. In each case excision of the wound and air tight closure of deep tissues and skin gives rapid relief. Deeply placed silkworm sutures including skin, muscle and deep fascia, should be used. A moist dressing is applied or a dressing of gauze impregnated with paraffin emulsion. If there is a tension pneumothorax, a short wide bore needle is inserted into the pleura through the second intercostal space, 2 in. from the margin of the sternum. If necessary the needle is left in position and connected with a water seal bottle.

TREATMENT BEGUN LATER THAN TWENTY-FOUR HOURS AFTER INJURY—INCISION AND DRAINAGE

If more than about twenty four hours have elapsed since the injury was sustained, infection is already established in cellular tissues, fascial planes and lymphatics. Excision of the wound cannot prevent infection. On the contrary, a searching exploration for foreign bodies may open fresh tissue planes and disseminate infection more widely. The keynote of operation must be incision, not excision (Figs 371-373). The skin wound is enlarged, deep fascia is divided, muscles are separated in intermuscular planes and bone is drained exactly as in the operative treatment of osteomyelitis. Foreign

bodies which are easily accessible should be removed but the object of the procedure is to drain the wound not to explore it. Every undermining track is laid open by incision of overlying tissues. The surgeon should use his finger to explore the track in order to be certain that no recess, cleft or pocket remains in which discharges can accumulate and decompose. Digital palpation is recommended despite the requirements of no touch technique that a finger should never be put into the wound unnecessarily. The importance of complete saucerisation is so great as to override the objection. Rubber drainage tubes should not be used. Not only may they cause secondary hemorrhage by pressure on the walls of blood vessels but they should be quite unnecessary. The wound should be so freely incised in the



FIG 371

Wrong way of using the closed plaster technique

Infection had been introduced by three short incisions with long strips of rubber dam placed into the intercommunicating tracks. The limb was then enclosed in plaster. When the plaster was removed and the rubber strips withdrawn pus poured out from a continuous access extending from ankle to knee. The rubber dam had been placed over the incisions rather than draining the infection (see Fig 374).

long axis of the limb that the insertion of a rubber tube is impossible because it falls out. The surgeon who insists on using short incisions and tubes or rubber dam strips must not employ the closed plaster method. The short incision is always unwise but when enclosed in plaster it is dangerous. It is unwise because a 10 in incision heals just as quickly as a 2 in incision. It is dangerous under plaster because the rubber dam so often plugs the drainage wound and causes spreading infection (Figs 371-374).

When the wound has been freely laid open vaseline gauze is lightly and gently placed in the cavity so that the walls are kept apart. This is usually described as packing the wound but the word pack is unfortunate because it gives a false impression of tight plugging. Actually the gauze is inserted quite loosely. The nature of the pack is unimportant as compared with the

operation which precedes its insertion. Vaseline gauze¹ is recommended because it is non irritant and the even pressure of its mass prevents œdema and excess granulation. Serum and pus track to the surface between the granulations and the pack which is gradually pushed out as the wound heals from the bottom. Trueta² recommends plain gauze and an unpadded plaster without skin protection believing that discharge is sucked out of the wound through the gauze into the plaster. Such capillary action, if it exists at all, must cease within a few hours. Moreover plain gauze tends to become enmeshed by the granulations and unprotected skin rapidly develops a purulent dermatitis. It is better to protect all skin over which pus will flow



FIG 372



FIG 373

Correct way of using the closed plaster technique

Same case as Fig. 372 after the infected wound had been correctly incised, drained and lightly packed with gauze. The wound is now saucerised and can be enclosed in plaster with safety (see Figs. 374-378).

by a layer of vaseline gauze. Other surgeons advise that the wound should be packed with flanne gauze, gauze soaked in hypertonic saline, saturated sodium sulphate, petroleum jelly³ and cod liver oil⁴.

If there is a compound fracture, displacement of the fractured bone is then corrected by manipulation exactly as if it was a closed injury and a complete plaster is applied. No window is cut because the wound has already been drained, inspection is unnecessary and repeated dressing is harmful.

¹ Vaseline gauze is prepared by placing successive layers of gauze folded to three or four thicknesses (or wide rolls of gauze) in a tin box into which is placed sufficient vaseline to cover the gauze, until the vaseline is melted. The tin is then autoclave for not less than 15 minutes. It should be sterilised once in 15 days. Each time it is used. Tull gauze is a similar preparation. A red curtain is hung with a mesh of 1/2 inch cut 1 to 2 cm. squares, placed in a tin box with grease proof paper between each set, are of 1/2 inch and 1/2 inch box then fill it with a mixture of 80 parts paraffin (80 gm) 10 parts of ceru (20 gm), melt it all (20 gm) is sufficient to impregnate and cover the material after sterilisation. Squares of tull gauze of these dimensions are suitable for dressings after skin grafts.

² J. Trueta. Closed Treatment of War Fractures. *Lancet* 1939 I 1152, and II 1173.

³ S. M. Lichtenstein. War Wounds of the Limbs. *Lancet* 1940 II 31.

⁴ M. Lichtenstein. Cod Liver Oil Dressings—their Mode of Action. *Lancet* 1939 II 1073 (with 11111 graphs).



FIG 374
S. T. 24th August 1941



FIG 375



FIG 376
S. T. 24th August 1941

Closed plaster technique for infected wounds

If infection spreading from foot to leg (cf. Fig. 378) can be an excuse for a delay in the use of plaster, extension of the plaster to the ankle is inadequate drainage of the three short incisions (Fig. 374). The skin is subjected to constant pressure from the plaster of the abdomen (Fig. 375) the cavity of the plaster is a closed space (Fig. 376) and the incision is closed by the plaster.



FIG 377
Sgt T, 20th September 1942



FIG 378
Sgt T, 12th November 1942
Closed plaster technique for infected wounds

One month later (Fig 377) the wound looks gruesome but it is now lined with healthy granulations and not necrotic tissue moreover its size is due to elastic retraction of skin (not destruction of skin) and it will therefore pull together rapidly now that infection is controlled. At the next change of plaster only seven weeks later (Fig 378) the wound is almost completely healed - it was perfectly healed when the last plaster was removed in December

AFTER TREATMENT OF CONTAMINATED AND INFECTED WOUNDS BY THE CLOSED PLASTER METHOD

The practice of draining an infected wound by wide incision, enclosing it in suitable dressing, and refraining from meddlesome interference was practised many years ago by Gamgee. It was developed as the closed plaster treatment of infected fractures and osteomyelitis by Winnett Orr¹ in 1914-18. Since then it has been used routinely by many surgeons² and its merit is well established. It was finally developed in the Spanish War by Trueta³ who proved the value of the method in treating excised wounds even before infection was established.

The principles of treatment are (1) continued drainage without dressing until the wound is healed and (2) continued immobilisation without disturbance until the fracture is united. The plaster soon becomes stained with blood and purulent exudate, pus may track over the skin and be discharged from the ends of the cast, and a foul smell develops. These are not indications for changing the plaster. If there is no fever and no toxic reaction the wound must be healing.

Control of smell—The unpleasant smell of accumulating discharges may be masked by applying iodoform, mercurochrome, eucalyptus or creosote to the cast, or by sealing it with viscopaste at each end and latex paint and cellulose over its surface. The simplest and best method of controlling smell is to enclose the whole limb and plaster in a bag constructed of cloth with deodorising properties, such as the material known as "filter cloth—medical".

Change of plaster—After five or six weeks an anæsthetic is given while the plaster is changed and the wound dressed. Full aseptic precautions and no touch technique should be used, and care must be taken to avoid movement of the bone fragments. The wound will be filling from the bottom with healthy granulations which have partly pushed out the pick. Fragments of sequestered bone which are visible in radiographs or presenting in the wound are removed and vaseline gauze is again picked lightly into the cavity. The skin is gently cleaned with ether and protected once more with vaseline gauze. A new plaster cast is applied. Subsequent changes of plaster are continued at six or eight weekly intervals until the wound is healed and the fracture united.

Skeletal traction and wedging of plaster—During the first few weeks, repair of the fracture is so delayed by infection that the position of the fragments is relatively unimportant, and even if reduction is imperfect it is better not to change the plaster or to wedge it with the object of securing final adjustment of alignment and apposition. On the other hand, after five or six weeks when infection is controlled and hyperæmia is subsiding granulation tissue grows and calcifies with increasing rapidity, and accurate reduction is essential. When the plaster is changed if the fragments are still overriding skeletal traction should be used exactly as in closed fractures, infection of the pin track being avoided by cleaning the skin thoroughly and sealing the punctured wounds with collodion. Check radiographs are taken the next day, and if alignment of the fragments is not perfect, the angulation must be corrected by wedging the plaster (p. 174).

¹ Winnett Orr. Osteomyelitis and Compound Fractures. London 1909.
² Watson Jones. Modern Treatment of Bone Infections & Compound Fractures. Brit Med J 1935 II 1015.
³ J. Trueta. Treatment of War Wounds and Fractures. London 1919.

sequestrum consists of the whole thickness of the shaft of a long bone its removal should be deferred until sufficient bone has been laid down as an involucrum to prevent collapse of the periosteal tube. It is the same principle of treatment which applies to the total excision of all bone fragments at the time of wound excision and to the treatment of acute osteomyelitis by

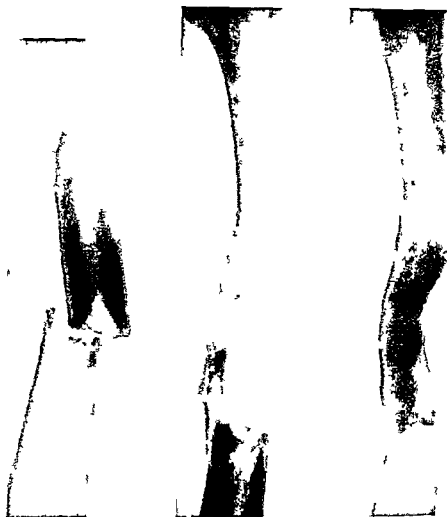


FIG 389

FIG 390

FIG 391

Closed plaster technique after massive sequestrectomy

Sequestrum consisting of 4 in. of the shaft of the femur (Fig 389) removed and an involucrum had formed so that continuity of bone was maintained (Fig 390) and if possible unruptured in obliteration until sound union without shortening had taken place (Fig 391). The patient was a plaster and he received a full fly netting (see p. 383).

diaphysectomy. Any operation which allows collapse of the periosteal tube and obliteration of the subperiosteal hematoma is likely to cause non-union with a wide gap between the fragments often necessitating a difficult bone grafting operation and sometimes causing serious shortening of the limb. It is not necessary to wait for the involucrum to be complete and for the bone to unite round the sequestrum. This might well involve a delay of

twelve months or two years. As soon as subperiosteal ossification is evident in the radiographs the operation can be performed.

Even when it is necessary to remove 4 or 5 in. of the shaft of the femur sound union of the fracture can be secured without shortening provided that special care is taken to immobilise the region continuously and without interruption (Figs 379-383). It is particularly important to maintain this continued protection throughout the time of operation and of every subsequent redressing or replaster. A single careless movement breaks the soft callus and if movement is repeated whenever the plaster is changed the



FIG. 38



FIG. 381

Closed plaster technique after massive sequestrectomy

Same case as Figs 379-381. Fig. 38 shows the condition at the first change of plaster one month after sequestrectomy. Fig. 381 is at the second change of plaster and the noticeable lateral large wound is due to retraction of skin (without destruction of skin) healed rapidly under plaster as soon as infection is controlled.

fracture will never unite. Before operation the patient is fixed in position on a traction table with a strong pull from a skeletal pin in the bone to the traction screws of the table. The site of fracture is supported to prevent even momentary backward sagging. While traction and fixation is maintained the sequestrum is removed, the wound lightly packed with vaseline gauze and a double plaster spica applied incorporating the traction pin. Two or three months later when the plaster is to be changed the skeletal pin is again fixed to the traction table and the thigh is supported while the plaster is changed. It may be necessary to continue the protection uninterruptedly for six months or longer but if care is taken to avoid any single movement the fracture will unite.

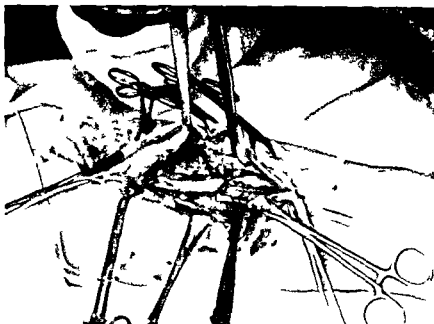


FIG. 384



FIG. 385

Closed plaster technique after sequestrectomy

Compound fracture of the tibia with sequestration of a large part of the shaft (Fig. 384). The operation is performed by soft tissue dissection along the shaft, scraping and exposing the bone. The ends of the bone are kept slightly apart by loose packing with vaseline gauze. The fracture is

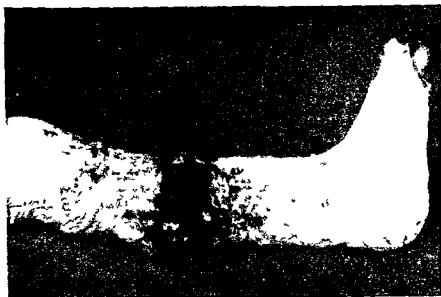


FIG. 380



FIG. 381

Closed plaster technique after sequestrectomy

The plaster is heavily stained and there is often a foul smell (Fig. 380). Nevertheless the wound is healing beneath the plaster. Six weeks after operation at the first change of plaster the wound is almost completely healed (Fig. 381). Plaster immobilization continues until the fracture is united.

PLASTIC SURGERY IN WOUNDS AND COMPOUND FRACTURES

The closed plaster treatment of wounds and compound fractures is of inestimable value in controlling infection assisting the union of fractures avoiding interference with the growth of bone and promoting the growth of granulation tissue. But the closed plaster treatment does not promote the growth of epithelium. Indeed the flow of pus over the surface of a limb which is not protected by vaseline or petroleum jelly causes digestion even of normal skin. It may give rise to extensive ulceration. Similarly the growth of epithelium over a granulating wound is hindered rather than promoted by bathing in pus and enclosure in plaster. Pus is detrimental to epithelial growth.

A clear distinction must be drawn between large wounds due to the gaping of incised skin and large wounds due to the destruction of skin. A wound produced by incision of an abscess or infected bone which is picked open with gauze may measure 10 in in length by 3 or 4 in in width. Nevertheless the wound heals rapidly as soon as infection is controlled even when it is covered with plaster. It may be healed completely within about six weeks not because new epithelium has grown but because elastic contraction of tissues and firm support of plaster have encouraged approximation of the gaping margins of the wound (compare Fig 377 with Fig 378 and also Fig 382 with Fig 383). On the other hand if skin has been destroyed over an area 10 in by 4 in healing can take place only by the growth of new epithelium. For from the wound being healed in six weeks many months will now elapse and healing will be delayed still longer if the wound is enclosed in plaster and bathed in pus. It is clear therefore that when skin has not been destroyed treatment by the closed plaster technique may be continued to a conclusion. By the time infection is healed and the cavity is filled with granulations the skin margins will be approximated and healing complete. But if skin has been destroyed it is unwise to persist with the closed plaster method after the cavity is filled with granulations and in the case of compound fractures after recalcification of bone and union of the fracture. Epithelialisation can be secured more rapidly by other methods. Rapid growth of epithelium is of great importance because without it a vicious circle may be set up which prevents the wound from healing at all.¹ In normal healing granulation tissue and epithelial tissue grow simultaneously. As the wound becomes older young granulation tissue is replaced by vascular fibrous tissue. If the growth of epithelium has not kept pace with the development of mature fibrous tissue the gradually diminishing blood supply produces an increasingly unfavourable base for the spread of epithelium.² Infection recurs epithelial growth is still further impeded more fibrous tissue is deposited and the cycle repeats itself indefinitely. In some regions notably over the subcutaneous surface of the tibia where the underlying base is bone and not muscle so that there is no assistance by elastic contraction of the wound early delay in epithelialisation may give rise to a chronic ulcer which persists for months or years. The destroyed skin should be replaced by skin grafting before

¹ J. M. C. Versa. *Orthopaedic Aspects of the Leg Fracture*. *Proc Roy Soc Med* 1941 xxxi 791.
² G. R. Morley. *Co-operation between the Bone and Orthopaedic Surgery*. *Proc Roy Soc Med* 1941 xxxi 760.

this vicious circle is set up preferably at the time of wound excision or at the latest at the time of the first change of plaster. Only in neglected cases is skin grafting required after union of the fracture.

Skin grafting at the time of wound excision—The best dressing for an

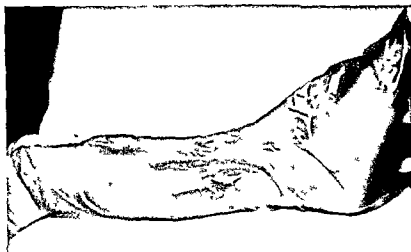


FIG. 388



FIG. 389

Bunyan bag technique

When skin is destroyed rapid healing does not occur under plaster and skin grafting is necessary. Infection must first be controlled by antiseptic irrigation. A Bunyan bag is sometimes useful. (Treated by Wing Commander B. H. H.)

excised wound is skin and if skin has been destroyed the ideal time for its replacement is the time of the primary operation. The conditions of healing then approximate to normal and the wound heals without fibrosis or contracture. A thick razor graft ('intermediate' or 'split' graft) is cut from the medial or lateral aspects of the thigh with an ordinary knife with



FIG 390



FIG 391

Technique of split skin grafting

The granulation tissue layer is exposed (Fig 390). A split skin graft is cut from the thigh with a Humby knife (Fig 391).



FIG. 392



FIG. 393

Technique of split skin grafting

The skin graft is spread on tulle gras. Sulphamylate powder is massaged into the wound (Fig. 391). Firm pressure is applied by crepe or gauze bandaging over many layers of wool

razor sharp edge or more easily with a Humby knife and laid directly on the excised tissue. Such grafts take very well on muscle cellular tissue or periosteum but they seldom take on bone which has been deprived of its periosteum or on tendon deprived of its sheath. Sometimes large avulsed flaps may be sutured back into position with safety but when the base of the flap is not sufficiently wide to ensure an adequate blood supply the flap should be denuded of all fat until the base of the cornu is exposed so that it is converted into a full thickness skin graft with the slight advantage over a free graft of its attached base and limited blood supply.

Skin grafting over granulations—If the opportunity for replacing devolved skin at the time of the original wound excision has been missed a skin graft may be applied to the granulating wound at any later date. The important



FIG. 334

Pedicle skin grafting

Ulcers over bare bone or tendon need a full thickness graft transferred as a pedicle flap from the opposite calf. The flap must not be more than half as long as it is broad.

conditions for success are freedom from infection which destroys the skin and firm fixation of the graft to its bed. **Control of infection**—Staphylococcal infection is relatively harmless but skin grafting is seldom successful in the presence of the hemolytic streptococcus. Even apart from the danger of lifting of the graft by inflammatory exudate grafts which at first appear to be successful often undergo late ulceration and destruction. B pyocyanus may also cause failure the graft becoming thick and greasy in appearance and finally being removed as a slough. Three measures are available by which to control infection (1) when there is no fracture gross infection may be controlled by saline or hypochlorite irrigation the Bunyan lag technique is useful because the wound is irrigated at intervals without exposure and trauma of the granulating surface (Figs 388-389). (2) as the first step of the operation granulations are shaved off with a sharp scalpel or razor this excision of infected granulations down to the fibrous tissue by eliminating the pus producing tissue increases the percentage take of

skin grafts from little over 0 per cent to nearly 90 per cent (3) after excision of the granulations and control of hemorrhage by firm pressure with adrenaline packs sulphamidamide powder is gently massaged into the wound base. Chemotherapeutic control of the hemolytic streptococcus has marked a great advance in the success of skin grafting operations. *Pressure of the graft on its bed*—The second factor by which success is determined is firm pressure of the graft on to its bed. The graft must not be lifted by the exudate of infection or by hemorrhage. This is achieved (and general handling of the graft assisted) by spreading it over skin surface down on a layer of tulle gras or vaseline gauze which is then turned over and firmly pressed on to the prepared bed. Pressure is maintained by the firm application of alternate layers of wool and crepe bandage. If the wound is associated with a compound fracture plaster is then applied. It is wise to change the plaster at an earlier date than would otherwise be arranged. After ten days the wound is cleaned of exudate and pus; it is powdered with sulphamidamide; firm pressure is reapplied and the limb is replastered.

Late skin grafting—In late cases a split skin graft may still be used but when the wound lies over exposed tendons or the subcutaneous surface of the tibia a direct transference whole thickness flap is often necessary. To cover a defect over the tibia a flap is cut from the opposite calf left attached by its base (which must be at least twice as great as its width) and stitched in position over the excised area. The two legs are fixed by means of plaster in such a position that there is no strain on the graft (Fig. 394). The base of the flap is cut from the normal leg after three or four weeks (or longer if the circulation of the flap is in doubt or if exudate has tended to raise the flap from its bed). The raw area on the opposite calf is covered with a razor graft from the thigh. In wounds of the upper limb a whole thickness graft may be transferred directly from the abdomen or chest.

Skin grafting in delayed union of compound fractures—The healing of a chronic ulcer in the region of a fracture has the further advantage of accelerating delayed union. Recalcification of callus is delayed by infection and infection may be maintained no less certainly by an unhealed ulcer than by a sequestrum or bone abscess. This acceleration of union is sometimes very striking in old compound fractures of the tibia. Union which has been delayed for many months is often consolidated within a week or two of completing the skin grafting operation.

OTHER APPLICATIONS OF THE CLOSED PLASTER TECHNIQUE

"Degloved" extremities—If skin is torn from a limb a vaseline gauze plaster may be applied in order to prevent the development of deformity while new epithelium grows or skin is replaced by grafting. A man of forty-two sustained severe laceration of the foot with avulsion of the four outer toes and the whole skin thickness of the dorsum of the foot. It had been proposed to amputate the foot but the great toe and its metatarsal were uninjured. The wound was excised, a closed plaster applied with the foot in right angled dorsiflexion and after two weeks skin grafts were applied to the dorsum of the foot. Closed plasters were reapplied at intervals until the wound was healed. Although the man now has less than half a foot he has the great toe which is the most important part. There is no contracture.



FIG 39



FIG 39b

Burns and fractures of the same limbs

When there are burns and fractures of the same limbs the closed plaster method is often essential. This patient has a fracture of the shaft of the femur and of the head of the tibia as well as severe burns of face, arms, hands and legs. The closed plaster method is often advisable for burns even when there is no fracture.

or deformity, he walks with a normal heel and toe gait, and has done heavy labouring work for eight years with the loss of only one week owing to temporary ulceration of the skin

Severe pressure sores—Large sloughing bed sores over the sacrum, iliac crests or other bony prominences can be treated safely beneath a closed plaster, thus permitting continued immobilisation of fractures, dislocations or infected joints. If vaseline gauze is applied and care is taken to mould the plaster smoothly the pressure sore will not increase but will slowly heal. The closed plaster treatment is not, however, advocated for pressure sores unless immobilisation in plaster is needed for some other reason, because more rapid epithelialisation can be secured by other methods and particularly by skin grafting

Burns and fractures of the same limb—Pilots and other members of air crews often sustain burns and fractures of the same limb (Figs 395-396). Vaseline gauze is applied to the burnt area, and the limb is treated by manipulation and plaster exactly as for a closed fracture. Even deep burns of the dermis and whole thickness of the skin heal perfectly beneath the vaseline gauze of a closed plaster. The technique may also be indicated when there is no fracture. If the healing of a burn threatens to produce joint contracture, the limb may be immobilised in plaster in the corrected position, and the granulating surface will heal without contraction of the scar. Moreover, skin grafts may be applied and covered with vaseline gauze or 'tulle gras' and the growth of new epithelium continues despite enclosure in plaster

Infected amputation stumps—It is often advisable to treat an infected amputation stump by light packing of the wound with vaseline gauze and enclosure of the whole stump and the proximal joint in plaster. The immobilisation controls infection and relieves pain, the support accelerates healing of the wound and the plaster prevents flexion deformity of the stump. If there is fear of secondary hæmorrhage a small window may be cut in the plaster over the main artery, so that bleeding can be controlled while the plaster is removed and the wound explored (Fig 403). The closed plaster treatment is particularly useful in the hand when several fingers are destroyed. If only one digit remains uninjured, whether it is a finger or a thumb, every effort must be made to provide a stump against which the active digit can be opposed. If, as in the case shown in Figs 397-399, the thumb is not seriously injured, one finger should be preserved even if it is completely shattered and its tendons totally destroyed. Provided that the blood supply is adequate, the shattered finger is enclosed in vaseline gauze and plaster and fixed in a suitable position opposed to the thumb. The resulting stump will be invaluable. Similarly, a thumb with intact blood supply should always be preserved even if bones are gravely comminuted, joints injured beyond hope of useful movement, tendons destroyed or even skin avulsed. Skin can always be replaced by grafting, damaged bones and joints heal beneath a closed plaster, and even if the stump is completely rigid, it will more than double the usefulness of any mobile finger which remains. Even if all fingers are lost, it is worth preserving a thumb and arranging later for the fitting of a prosthesis against which the thumb can grip (Figs 400-402).



Fig. 397



Fig. 398



Fig. 399

Explosive wound of hand. Three fingers were blown off. The fifth finger was shattered and seriously infected, but by the closed plaster technique it was preserved. It serves as an invaluable stump in opposition to the uninjured thumb. (Mr. S. Cohen & Co.)



Fig. 400



Fig. 401



Fig. 402

Closed plaster technique after amputation

An incendiary dropped from a bomber crashed through one of our aircraft below and to strike an observer's hand. (The actual bomb is shown in Fig. 403.) Only the thumb could be saved.

The thumb was preserved by the closed plaster method. (Operated on by H. A. C. Maudslayi, M.D., F.R.C.S.)

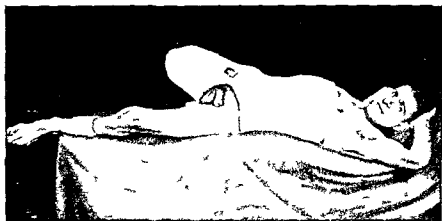


FIG 403

Infected amputation stump treated by closed plaster method The window has been cut in case of secondary hæmorrhage



FIG 404



FIG 405



FIG 406



FIG 407



FIG 408

Closed plaster technique after amputation

Gangrene treated in closed plaster until the demarcation line formed. Note the cheerful face of the patient despite the gangrenous limb (Fig 405). By continuing the closed plaster treatment after circular amputation it was possible to save a below-elbow stump (Fig 408). (Bye artery f.v.s. (den))

intravenously or given in a continuous drip saline. For prophylaxis 2 gm should be given as soon as possible after injury, followed by 0.5 gm doses four hourly for a period of from seven to fourteen days. If infection is already established the dose should be increased to 1 gm four hourly for two days and then reduced gradually as the clinical condition improves. It should seldom if ever be necessary to give more than 35 gm.

Local administration may also be used the drugs being applied to the wound in the form of a powder or as tablets which are absorbed more slowly. The advantage of local application is that bacterial growth in shed blood and blood fluids is inhibited while at the same time the drug is absorbed into the blood stream and so reaches bacteria which have already invaded the tissues. The treatment is of value in the prophylactic chemotherapy of excised wounds which have been sutured (p. 214) but local applications are worthless in drained infected wounds because the bacteriostatic action is completely inhibited by pus fluid.¹ Solutions of the sodium salt of sulphapyridine should not be applied to wounds. So great is the alkalinity of this preparation (pH 10) that it is injurious to all tissues and particularly to the tissues of the brain.

Dusting of granulations—The healing of granulating wounds can often be accelerated by the daily blowing of a fine dust of sulphonamide powder over the wound thus inhibiting the growth of streptococci and other bacteria.

Complications of chemotherapy—Agranulocytosis—If the clinical condition does not respond rapidly to chemotherapy the treatment should cease. Continued administration may cause agranulocytosis which has proved fatal in 50 per cent of recorded cases.^{2,4} The complication has seldom occurred before the second or third week of continuous treatment and in most cases more than 50 gm of sulphanilamide have been given. If treatment is continued for more than seven days or if more than 25 gm have been given white cell counts must be done at frequent intervals.⁵ A falling white cell count demands immediate cessation of chemotherapy, the intramuscular injection of pentonucleotide (0.35 gm twice daily) and blood transfusion.

Cyanosis—Cyanosis due to methæmoglobinæmia does not call for cessation of the treatment and need cause no alarm. It may be relieved by giving 0.5 gm methylene blue by mouth twice daily.

Other complications such as malaise vomiting acidosis drug fever dizziness headache dermatitis^{6,7} and hæmaturia are in themselves of little significance and if the white cell count is not falling the treatment may usually be continued.⁸

Penicillin,^{9,11} an extract from the growth of a mould is a much more powerful bacteriostatic agent than any of the sulphonamide drugs. It controls the growth of staphylococci as well as of streptococci and anaerobes and it is not inhibited by pus peptones or the presence of large numbers of bacteria.

¹ A Fleisling *Proc. Roy. Soc. Med.* 1940, xxxii, 197.

² L. Colebrook *Chemotherapy and the White Cells* *Lancet* 1939 ii, 138.

³ G. A. Scott and O. Meerapfel *The Effect of Sulphonamides on Blood Serum* *Lancet* 1939 ii, 144.

⁴ R. A. Coxon and J. R. Forbes *Agranulocytic Angina after M & B 693* *Lancet* 1934 ii, 141.

⁵ D. Campbell *Sulphanilamide Chemotherapy* *Lancet* 1939 ii, 371.

⁶ E. McKim *Dermatitis from Sulphonamide Compounds* *Brit. Med. Jour.* 1939, ii, 104.

⁷ R. Hallam *Severe Skin Reaction Following Administration of M & B 693* *Brit. Med. Jour.* 1939, i, 559.

⁸ Chemotherapeutic Agents for Septic Wounds—War Office Memorandum *Brit. Med. Jour.* Oct 21 1939 834.

⁹ A. Fleming *Antisepsis in War Time—Penicillin* *Brit. Med. Jour.* v, 1940, 15.

¹⁰ Chas. Florey and others *Penicillin as a Chemotherapeutic Agent* *Lancet* 1941, ii, 104 and 1941 ii, 1.

¹¹ D. Heiman and W. Herrell *Penicillin and Gramicidin* *Proc. Mayo Clin.* 1941, x, 131.

GAS GANGRENE AND TETANUS

The bacteria of gas gangrene and tetanus infections are anaerobic¹. Bacteriological examination of many wounds shows the presence of the causal organisms of gas gangrene (*Cl welchii*, *Cl septicus* and *Cl œdematiens*) but the infection cannot become established unless the tissues in which the bacteria have been implanted are anaerobic. This does not mean that the wound must necessarily be exposed to the external air or that oxygen must be supplied by hydrogen peroxide irrigations and dressing. Even more free oxygenation is available through the normal blood supply. If the walls and contents of a wound consist only of living pulsating tissue the conditions are aerobic and gas gangrene bacilli cannot survive. It is for this reason that the gas gangrene bacilli which normally inhabit the rectum do not infect the wounds of hæmorrhoid operations and that individuals who are careless in their morning toilet do not develop gas gangrene in skin abrasions of the fingers. It is for this reason also that a properly excised wound freely drained by a vaseline gauze pack and completely enclosed in a Winnett Orr plaster does not develop gas gangrene even although the external air is excluded. If on the other hand a wound contains foreign matter and necrotic debris and its walls consist of dead and dying tissue the conditions are anaerobic and gas gangrene infection can develop. Deep penetrating wounds with destruction and necrosis of muscle gunshot wounds of the avascular retroperitoneal cellular tissues and wounds contaminated with foreign bodies like pieces of clothing are therefore the most dangerous injuries. The infection is usually the result of faulty surgery—the wound was not excised early enough it was not excised adequately or it was sutured under tension when it should have been drained. Nevertheless even with perfect surgery it may not always be possible to achieve the conditions necessary to inhibit the growth of these organisms. Avascularity of the tissues may be the result of remote interference with the blood supply. Moreover the infection may be fully established within three or four hours of injury.

The clinical signs of gas gangrene—A rising pulse rate in a wounded man who has recovered from the initial shock and is not suffering from continued hæmorrhage is significant. There is pyrexia and the facial expression is anxious and alert. The local signs are pain swelling crepitation of tissues and a curiously indolent dry appearance of the wound but with a thin watery foul smelling exudate which escapes on gentle pressure. Only when the anaerobic infection is controlled does the discharge become a frank purulent exudate and herein lies the explanation of the laudable pus of an earlier generation of surgeons. The smell is characteristic once recognised it is never forgotten.

Three types of gas gangrene infection may be distinguished (1) local infection of cellular tissues in the neighbourhood of the wound (2) infection of one muscle or a group of muscles (3) massive gas gangrene of all muscles and cellular tissues of the limb.

Treatment of gas gangrene—Surgical measure treatment is essential. The wound must be so free of tension that every tissue will regain normal blood

¹ *B. (bact.)* *Gas (gangrene)* *a. (Gang.)* *Inf. (in)* *Joint*

must be removed. The wound is extended in the long axis of the limb until it is 10 or 12 in in length. One or more other incisions may be added on the opposite side of the limb. Every muscle which is gangrenous must be removed in its entirety, the utmost care being taken to avoid damage to the blood supply of adjacent muscles. All wounds are left widely open. If the infection is a massive gas gangrene, immediate amputation is essential, the flaps being left widely open until the gas gangrene infection is controlled. Secondary suture is usually safe after about ten days.

Chemotherapy and antitoxin therapy—The sulphonamides inhibit the growth of *Cl welchii* and *Cl septicum*¹ but not of *Cl oedematiens* whereas antitoxin therapy is most effective against infection with *Cl oedematiens*. Both forms of treatment should therefore be used. Sulphathiazole appears to give better results than sulphapyridine or sulphamidamide. Polyvalent antitoxin is given intravenously (3 ampoules as now dispensed totalling 27 000 units *Cl welchii* antitoxin, 13 500 units *Cl septicum* antitoxin, 9 000 units *Cl oedematiens* antitoxin) and repeated as necessary while the toxæmia persists^{2,3}. The risk of anaphylactic reaction is small, but a syringe charged with 1:1000 adrenaline hydrochloride should be kept ready for injection.

X ray therapy—Recent reports indicate the possible value of radiotherapy in gas gangrene infections which are not amenable to radical surgical treatment.^{4,6} A small dose is delivered at frequent intervals over a short period (for example 75 r to each of two fields twice daily for three days).

Tetanus infection—The anaerobic spore forming *Cl tetani* develop under similar conditions to those which govern the growth of gas gangrene bacilli. Three degrees of infection may be distinguished: (1) local rigidity and twitching in the region of the wound; (2) generalised tonic rigidity; (3) recurrent reflex spasms. When the infection is fully developed trismus, dysphagia, risus sardonicus, opisthotonus and abdominal rigidity present a clear clinical picture. If reflex spasms develop within twenty-four hours the prognosis is grave; if within two or three days the prognosis is fair and if not until after the fifth day the outlook is good.

Prophylactic treatment—The development of tetanus infection can be prevented or greatly modified by early excision of the wound and by the prophylactic injection of antitoxin given immediately after the infliction of a wound. 3000 international units must be injected as soon as possible. The immunity lasts only two or three weeks and the injection may need to be repeated especially if it is proposed to remanipulate a compound fracture or reopen a wound. Many volunteers in the military services have already been protected by the active immunisation of tetanus toxoid injections, but there should be no hesitation in giving prophylactic antitoxin treatment even if it is believed that a wounded man may previously have received toxoid injections.

Treatment of established tetanus infection—If tetanus infection is established, 200 000 international units of tetanus antitoxin may be injected intravenously at the first possible moment. A further 50 000 units should

¹ Bohlman: "Gas Gangrene Treated with Sulfanilic Acid," *J. Amer. Med. Ass.* 1937, clix, 9, 4.

² Lower and Tormey: "Gas Gangrene and Its Treatment," *Surg. Clin. N. Amer.* 1937, xvij, 1385.

³ No report on Treatment of Gas Gangrene. *M. J. Brit. Resear. Council War Med. J.* 1943, vi, 1, 11-15, 1943.

⁴ J. F. Kelly: "Radiology," 1934, xx, 296; 1935, xxxi, 695; 1939, xxxix, 6, 5.

⁵ R. L. Sewell: "Surgery," 1929, vi, 1, 1; *Lancet*, 1940, i, 900.

⁶ J. F. Brailford: *Brit. Med. J.* 1940, i, Feb 17, 1940, 24.

be given every seven days until the reflex spasms subside. There is probably no advantage in giving antitoxin by the intrathecal route. Reflex spasms may be controlled with avertin or paraldehyde. No manipulation or operation should be performed on the wound until a full dose of antitoxin has been injected and given time to circulate. Even after the lapse of many months or years spores which remained dormant in the bone may be lighted up¹ and late operations must also be preceded by prophylactic antitoxin therapy.

AMPUTATIONS FOR OPEN AND INFECTED FRACTURES

Indications for immediate amputation—Amputation is seldom necessary in the treatment of bone and joint injuries and the decision to adopt this unusual and drastic measure should always be supported by a second opinion. If the main blood vessels are not destroyed and there is no irrecoverable nerve lesion the limb should be saved no matter how severe the contamination of a wound, the comminution of a fracture or the destruction of skin. Even serious infection may be controlled by the closed plaster technique. Skin destruction over the whole circumference of a limb can be treated successfully by skin grafting and if immobilisation is continuous and prolonged contaminated wounds heal and shattered bones unite. Almost the only indication for immediate amputation is the destruction of main blood vessels and the imminence of gangrene.

Technique of immediate amputation—Within about eight hours of injury bacteria have not invaded deep tissues and lymphatics and infection is not established. If the wound is remote from the site of amputation so that all dead and contaminated tissue will be removed and it is reasonably certain that the patient will remain under observation for several days after operation a formal flap amputation may be performed at the site of election with immediate suture of the skin flaps. If more than about eight hours have elapsed if the amputation must be performed through wounded tissues or if early evacuation of the patient may become necessary delayed primary or secondary skin suture must be employed as in the technique of late amputation.

The site of election and the minimal length of stumps is illustrated in Figs. 409-412.* The ideal stump is firm conical in shape so that it will fit the bucket of an artificial limb and free from skin folds which cause intertrigo, eczema. The flaps should therefore be circular and their combined lengths must be exactly equal to the diameter of the limb. Cutaneous nerves of the digits and thigh should be shortened slightly but the main nerves of the limb must not be pulled down, crushed, ligated or injected with alcohol.² The formation of a nerve bulb cannot be avoided³ and its best situation is near the end of the stump where there will be no pressure. Every bleeding vessel is ligated so that hæmorrhage is complete and drainage unnecessary. Stripping of periosteum or the cutting of periosteal flaps is of no value. Bone spurs form only on the posterior aspect of the femur and give rise to no symptoms. The stump should be left at rest for two or three weeks because early handling increases the tendency

* V. Bonney, C. Box and J. McLennan. Tetanus Bacilli recovered from Scar Ten Years after Post-operative Tetanus. *Brit. Med. Jour.* July 6, 1934, 10.

¹ Ministry of Health. Artificial Limbs and Relation to Amputation. H.M. Stationery Office, London, 1939.

² J. Verall. Amputation Stumps and Artificial Limbs. *Proc. Med. Soc. Jan. 13, 1910, 6.*

³ Max Inge. Amputations under War Conditions. *Brit. Med. Jour.* July 8, 1933, 7.

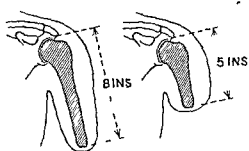


FIG 400

Optimal and minimal lengths
of upper arm amputation stump

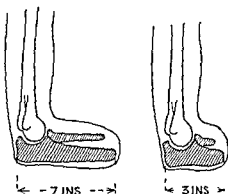


FIG 410

Optimal and minimal lengths
of forearm amputation stump

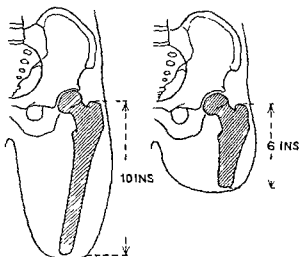


FIG 411

Optimal and minimal lengths of thigh
amputation stump

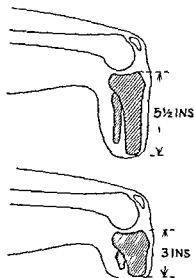


FIG 412

Optimal and minimal lengths of
below knee amputation stump

Over 40 000 amputations were performed in this country during the last war and the few years immediately following it. Since then tremendous strides have been made in the surgery of wounds and compound fractures and amputation is becoming increasingly rare. For example in one large series of casualties treated in R A F Base Hospitals the incidence of secondary amputation for spreading infection, gas gangrene, secondary haemorrhage and other complications was as low as 0.1 per cent despite a high proportion of severely infected and grossly contaminated wounds and compound fractures.

For further details of the technique and management of amputations the reader is referred to an excellent monograph 'Amputations and Artificial Limbs' by Langfale Kelham and George Perkins. Oxford War Manuals. London 1942.

to phantom limb Firm bandaging is then necessary in order to promote shrinkage of the muscles Fitting of the artificial limb should be possible within two or three months of amputation

Indications for late amputation—Toxæmia from spreading infection, repeated and dangerous secondary hæmorrhages, gangrene due to vascular thrombosis or massive gas gangrene may necessitate late amputation Infection is then established in deep tissues and has spread proximally along lymphatics far beyond the limits of the wound Even if the amputation is performed many inches above the level of injury if the wound is completely screened and the skin thoroughly prepared, the infection which has spread along lymphatic channels cannot be avoided, and infection of the amputation stump is almost inevitable Primary skin suture is unsafe and may be disastrous

Technique of late amputation—Two alternatives are available (1) flap amputation with delayed skin suture after from seven to ten days, (2) circular sleeve or 'guillotine' amputation at a low level with a formal flap amputation at the site of election after two or three weeks

Flap amputation with delayed suture—This is the operation of choice if the amputation must be performed at or above the site of election and there is viable skin below that level (as in the case of gas gangrene) Flaps are cut but not sutured Mattress silk-worm sutures through the skin flap margins are protected with rubber tubing or buttons and light traction is applied After a week or ten days when the danger of spreading infection and gas gangrene is over, the traction is released and the sutures are tied

Circular or sleeve amputation¹—If the first amputation can be performed well below the site of election a circular or 'guillotine' amputation is the operation of choice It can be performed rapidly, with minimal exposure of tissues and with perfect drainage Skin traction must be applied within twenty-four hours The muscles of the thigh and leg are powerful and long bellied and continued retraction produces a conical stump with projecting bare bone a wide area of granulations skin several inches above the cut surface of bone and unnecessary shortening of the stump (Figs 413-414) Vaseline gauze is applied and the no dressings technique employed so that unnecessary pain and fluid loss are avoided A formal flap amputation is performed at the site of election after several weeks, when infection is quiescent Reamputation before the granulating surface is clean and dry, and before the stump is free from œdema causes recurrent infection and defeats the whole object of the guillotine amputation

If the first amputation must be performed at or above the site of election and there is no viable skin below that level, a guillotine amputation with continued skin traction is again the operation of choice If traction is continued until the granulating surface heals with a terminal scar, the stump may be satisfactory even without reamputation whereas a flap amputation which would necessarily be two or three inches higher would be too short

¹ In a circular amputation the skin is flayed at one level the muscles at a higher level and the bone at a still higher level A guillotine amputation is strictly one in which skin, muscle and bone are divided in exactly the same plane Nevertheless, the experienced surgeon usually attracts each layer slightly before dividing the vessel and the peripheral nerves and skin with his knife. In this case a sleeve perhaps better described as a sleeve and still better as a guillotine amputation



FIG 413

Guillotine amputation left for ten days without skin traction showing the rapid development of conical stump due to retraction of thigh muscles and skin, causing unnecessary shortening of the stump



FIG 414

Method of application of skin traction with balanced weights to prevent retraction of the skin and muscles after guillotine amputation (Note that the pulley should be at a slightly lower level and the stump should not be supported on a pillow otherwise there is danger of flexion deformity of the stump)

PART II

PATHOLOGICAL FRACTURES AND BIRTH
FRACTURES

CHAPTER VI

SPONTANEOUS AND PATHOLOGICAL FRACTURES

The treatment of fractures cannot be divorced from general orthopedic surgery and nowhere is the impossibility of a watertight compartment of traumatic surgery more clearly illustrated than in the subject of spontaneous and pathological fractures. A fracture may be the first manifestation of a bone cyst or tumour. Recurrent fractures from trivial injuries may lead to the recognition of an obscure skeletal disease. The course of bone disease may be modified by a fracture. Very commonly the treatment of a fracture must be modified in accordance with the disease. The surgeon who attempts to treat fractures without a full knowledge of the pathology of bones and joints is faced with insuperable difficulties.

The terms spontaneous and pathological fracture are sometimes used as synonyms. It is an advantage to distinguish two groups of fractures in which the traumatic factor is unimportant: (a) spontaneous fractures where the bone is normal and (b) pathological fractures where there is a pathological abnormality of the bone.

SPONTANEOUS FRACTURES

Although the bone is of normal structure and strength fractures are sustained in the course of ordinary activity. Muscle inco-ordination and fatigue are important etiological factors.

Fractures of *tabes dorsalis*—The degeneration of the posterior columns of the cord accounts for loss of joint sensation and of pain so that the tabetic patient is unaware of the normal danger signals. Moreover there is muscle inco-ordination and hypotonicity which prevent him from taking advantage of the warning even if he was aware of it. The awkward and unguarded gait may therefore be responsible for spontaneous fracture of the patella or even for fracture of the femur or tibia. At this early stage of the disease the bones are normal and fractures unite promptly. In the later stages when Charcot's joints are developing true pathological fractures may occur¹ (Fig 415). The bone is then abnormally dense and the increased calcification causes increased fragility (Fig 416). These fractures are slow in uniting and there may be non union.

Fractures of epilepsy—The violent muscle contractions of epilepsy may cause spontaneous dislocation of the shoulder joint which is sometimes recurrent (Chap XXII). The great tuberosity of the humerus may be avulsed by the supraspinatus and in senile or bed ridden patients whose bones are osteoporotic spontaneous fractures of the spine or of the neck of the femur are often sustained especially when the convulsions have been forcibly restrained. In recent years convulsions artificially induced by cardiazol have been used in the treatment of psychiatric disorders. It is said that cardiazol is the elixir of life to a hitherto doomed race. The price to



FIG 41b



FIG 41a

Charcot's disease

Fig 41a.—Charcot's hip with pathological fracture neck of femur, complete destruction of the bone and multiple loose body formation. Fig 41b.—Charcot's ankle. Pathological crush fracture of os calcis. The bone is hypercalcified and fragile.

be paid is fracture of one or more vertebrae in about two fifths of the patients so treated¹ Over thirty spontaneous fractures of the femoral neck have been reported The results of nailing the bone are satisfactory even if the epileptic fits recur after operation²

March fracture of the metatarsals—Fractures of the shafts of metatarsal bones sometimes occur during ordinary walking in normal healthy individuals The injury may be sustained by a soldier in the course of a route march when there is an element of muscle fatigue In other cases the symptoms develop so gradually that the patient cannot believe that he has suffered an injury The fracture may obviously be of several weeks duration when advice is first sought (see Chapter XXXIII)

Metatarsus atavicus a congenital shortening of the metatarsal bone of the great toe is often a contributory factor The first metatarso phalangeal joint lies at a more proximal level than normal it may even be in line with the necks of the second and third metatarsals (Fig 417) Normally the metatarso phalangeal joints of the toes are level and the whole row may be regarded as one joint at which movement occurs with each step as weight is transmitted to the toes In metatarsus atavicus the axis of movement crosses the necks of the central metatarsals As the patient steps forwards there is an angulating strain on these bones and the 150 lb of superimposed body weight is more than enough to fracture them Other types of architectural weakening of the forefoot³ hypermobility of the first metatarsal adduction of the first metatarsal and transverse flat foot may be contributory factors Continued weight bearing on the fractured bone causes dissemination of the fracture hematoma and excessive callus formation The patient may even complain of the lump rather than of pain A walking plaster should be applied until union is firm and the foot is then strapped for a few weeks Exercises are taught for the intrinsic muscles of the foot If symptoms recur and there is a short first metatarsal bone removal of the second and third metatarsal heads to restore the line of the metatarso phalangeal joints is justified

One case has been recorded of amputation of the foot for a march fracture which was mistaken for a sarcoma⁴ If sarcoma of the metatarsals

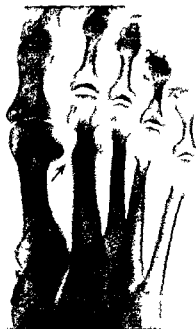


Fig 417

Spontaneous march fracture of second metatarsal already uniting when first seen with excessive callus from continued weight bearing

Palmer Vert. bral Fract. res. in Con. ul. ion Therapy *Lancet* 1939 ii 141

Gl. cane Blat. and Hank Fractures of Neck of Femur in Con. ul. ion Therapy *Lancet* 1940 i 450

* Bruce Anomalies of Forefoot in Relation to Metatarsal Displacements *Edin Med Jour* 1937 xlv 530

Dodd "Fixed Forefoot or March Foot" *Brit Jour Surg* 1934 xxi, 131

was not so rare as to be almost unknown the periosteal elevation and ossification and the absence of a history of injury might be confusing. Spontaneous fracture of a metatarsal bone must also be distinguished from Panner's disease—an allied disorder in which there is periosteal thickening of one or more central metatarsal shafts. In this condition there is impairment of the blood supply of the bone and usually avascular necrosis of the metatarsal head causing rigidity of the joint (Fig 418).

Fatigue fracture of the tibia—Crack fractures sustained as the result of ordinary weight bearing strain and without obvious injury may also occur in the tibia¹ (*fatigue fracture*) and in the neck of the femur.

Spontaneous dislocation of the toes—It is important to recognise that clawing of the toes in metatarsal flat foot especially when associated with

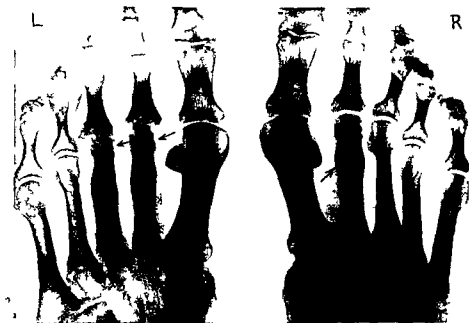


FIG 418

Panner's disease of second and third left and second right metatarsals. The left foot shows typical necrosis of the metatarsal heads. On the right there is only periosteal thickening suggesting a march fracture.

hallux valgus may progress to such a degree that the phalanx is pulled off the back of the metatarsal head by the tight extensor tendons. The metatarso-phalangeal joint is completely dislocated and the prominence of the metatarsal head in the sole causes severe pain until it is removed. One surgeon who operated on a bunion without recognising the coincident spontaneous dislocation of the second toe was threatened by medico-legal proceedings because the patient knew of no injury and blamed him for the dislocation.

PATHOLOGICAL FRACTURES

There are three groups of pathological lesions which may be responsible for weakening or destruction of bone and for pathological fracture.

(1) bone atrophy, osteoporosis and decalcification, (2) bone tumours and cysts and (3) congenital fragility of bone

Bone atrophy and decalcification—Prolonged recumbency debilitating and deficiency diseases senile change and excessive hyperæmia, cause bone atrophy and decalcification. The transverse trabeculae which are the last to be laid down in a growing child are the first to be removed in bone atrophy. This absorption brings the unchanged longitudinal trabeculae into relative prominence (Fig 426). The bone is weakened and it is susceptible to fracture. If the process continues the longitudinal trabeculae also undergo absorption the cortical bone is reduced to a mere shell and the bone buckles bends and breaks at many levels (Fig 423).

Bone cysts and tumours—Tumours of bone are the most common source of pathological fracture and the frequency is in direct proportion to the osteolytic and destructive qualities of the growth. This is borne out by the following analysis¹ of the incidence of pathological fracture in the more common bone tumours—

Multiple myeloma	69 per cent	Giant cell tumour	14 per cent
Cyst and cystic tumour	45	Osteogenic sarcoma	8
Secondary carcinoma	15	Ewing's sarcoma	5

Multiple myeloma is relatively rare so that the pathological fracture most commonly seen is that which occurs through a bone cyst and particularly through cysts and chondromas of the phalanges or metacarpals.

Congenital fragility of bone—Congenitally fragile bones are of two types. In fragilitas osseum the bone is slender porotic and fragile very like the bones of secondary atrophy. In Albers Schönberg disease the bones are dense solid and hypercalcified. Nevertheless they too are fragile because they are brittle. The term marble bones which is sometimes used is misleading. Chalk bones would be a better pseudonym.

BONE ATROPHY, OSTEOPOROSIS AND DECALCIFICATION

Disuse atrophy of bone—The disuse of prolonged recumbency or of immobilisation in splints or plaster causes decalcification and atrophy of bone. When activity is resumed care must be taken to avoid undue strains before recalcification is complete.

Poliomyelitis—In infantile paralysis there may be an extreme degree of skeletal decalcification especially in the early stages of recumbency and immobilisation. If a fracture is sustained it unites rapidly and normally.

Senile osteoporosis—Absorption of bone trabeculae and thinning of the cortical bone is a characteristic senile change and this type of osteoporosis is a common predisposing cause of fracture of the femoral neck. The spine is often involved and the pressure of intervertebral discs produces cupping of the vertebral bodies. The fracture shown in Fig 419 was sustained by an elderly doctor who merely stooped to lift a moderately heavy weight.

Hyperæmic decalcification—*Spontaneous dislocation of the atlas**—Decalcification of bone may be due to spreading hyperæmia from an adjacent inflammatory focus. Naso-pharyngeal and tonsillar infections can cause hyperæmic decalcification of the atlas sufficient to allow detachment of

¹ Geschlechter and Copeland. Tumours of Bone. New York 1936.

² Watson Jones. Spontaneous Hyperæmic Dislocation of Atlas. Proc Roy Soc Med., 1930, xxv, 526.
B & Jour Surg 1934, xxi, 46.

the transverse ligament. The anchorage of the atlas to the odontoid process is then impaired and it subluxates or dislocates forwards (Fig 420). As a rule the forward dislocation is incomplete and it is readily reduced. Perfect recovery follows immobilisation in plaster for two or three months. In some cases the cord is compressed and pathological dislocation may cause sudden death (Fig 421).

Infective decalcification—Pathological fracture in osteomyelitis—In acute osteomyelitis there is hyperæmic and infective decalcification of the bones



FIG 419

Senile osteoporosis

Pathological fracture of fourth lumbar vertebra.¹ There is general osteoporosis and collapse of the vertebral bodies from discompression.

and pathological fracture may occur unless the limb is adequately protected (Fig 422). The fracture must be treated like any infected compound fracture.

Rickets is due to a deficiency of vitamin D, the result of lack of fresh foods in the diet or lack of exposure to ultra violet light which synthesises vitamin D from the sterol in the skin. This vitamin promotes the absorption of calcium and phosphorus from the bowel. When it is lacking the skeleton becomes decalcified and porotic and is subject to bending deformities and pathological fracture. Fractures unite at an almost normal rate. The bones may be hardened by adding vitamins to the diet and by general open air treatment with exposure to sunshine.

¹ Moffat "Pathological Fractures of Spine with Disorders of Calcium Metabolism"



FIG 490



FIG 491

Spontaneous dislocation of atlas

The hyperæmia of tonsillar infection may decalcify the atlas and cause forward subluxation (Fig 490) or complete dislocation with paraplegia and sudden death (Fig 491 post mortem specimen)



FIG 492

Acute osteomyelitis

Pathological fracture in acute osteomyelitis of the femur. This case was immobilised in a Thomas splint and united firmly

Osteomalacia—In osteomalacia¹ and in famine malacia there is a dietetic deficiency comparable with the deficiency of rickets. It is often associated with pregnancy in a woman of twenty to forty, but the senile form occurs in both males and females. The blood serum calcium may be as low as 5 mg per cent and this distinguishes the disease from the somewhat similar bone changes of hyperparathyroidism (osteitis fibrosa) where the blood calcium is raised. The compact bone is absorbed until the cortex is of paper like thinness and the density of the radiographic shadow may be reduced almost to that of the soft parts (Fig 423). Multiple fractures and bending of the bones occur. Dietetic treatment is necessary with increased

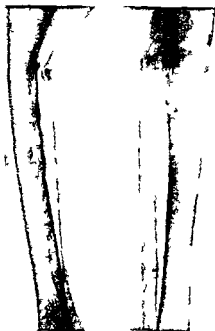


FIG 43

Osteomalacia

Pathological fractures of the fibula and tibia. Note the disappearance of primary and secondary trabeculae, thinning of the cortex and extreme osteoporosis.

calcium intake and the administration of ergosterol. In women affected during pregnancy the prognosis is good and there may be marked improvement after the menopause. In non puerperal types multiple fractures are associated with increasing pain and gross deformity and patients may become bedridden and die.

Celiac disease, steatorrhea and sprue²—In celiac disease of children idiopathic steatorrhea of adults and in tropical sprue there is imperfect digestion and absorption of fats with recurrent diarrhea. The stool is bulky and pale and chemical examination shows an excess of both split and unsplit fats. The resulting formation of calcium soaps and the generally accelerated flow through the intestine prevents the absorption of calcium and phosphorus. There may even be tetany. Severe osteoporosis with deformities and pathological fractures is the rule. So complete is the failure of

calcium absorption that union of the fractures is very slow unless the disease is vigorously treated. The fat intake must be reduced to 10 or 20 gm per day. Hydrochloric acid is given by mouth to relieve the associated achlorhydria and to promote calcium absorption and calcium gluconate is given by mouth or by injection. The fractures may require immobilization for many months.

"Idiopathic" osteoporosis of children—Children not suffering from celiac disease or rickets and with no evidence of osteitis fibrosa cystica due to parathyroid adenoma sometimes show very extreme generalized osteoporosis. There is loss of the secondary bone trabeculae, the bone cortex is thinned and multiple pathological fractures occur (Figs 424-427). The blood

¹ D. H. L. J. J. Surg. 1931 xv 71.
² O. Connally. The Known Factors. J. Med. Soc. 1911 17.
 Moore O. Farrell (Craig by Hayden and Moriarty). Celiac disease. Quart Jour Med. 1936 5 441.



FIG 424

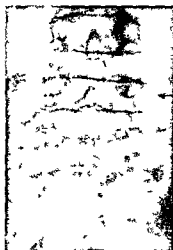


FIG 425



FIG 426



FIG 427

"Idiopathic" osteoporosis of children

Evidence of osteoporosis in the disappearance of secondary transverse trabeculae and in the thinning of the cortex (Fig 426). There has been a fracture of the ulna (Fig 427) of several dorsal vertebrae (Fig 424) and of the fifth lumbar vertebra (Fig 425).

calcium is normal but there is a loss of calcium balance, more calcium is excreted than is absorbed. Helfet¹ believes that this may be due to an error of phosphorus metabolism with a lack of balance between the phosphorus and the available calcium in the blood. A raising of blood phosphates it is thought may stimulate the parathyroid to physiological hyperactivity in order to mobilise calcium from the bones and form calcium phosphate which is excreted. Helfet has shown that if the phosphorus intake is reduced by giving alum acetate, which precipitates the phosphates in the intestinal tract, and at the same time calcium is given as milk or calcium gluconate, the calcium balance is restored to normal, and the bones gradually recalcify.

BONE CYSTS AND OSTEITIS FIBROSA

Solitary bone cyst²—The simplest type of osteitis fibrosa is the solitary bone cyst which occurs in children under fifteen years of age, usually in the upper shafts of the tibia, femur or humerus. In half the cases a pathological fracture is the first manifestation. The cyst is recognised radiographically by the bone expansion with a central defect which has symmetrical contours, a smooth unbroken margin, and no periosteal reaction (Fig. 428). The lesion is essentially benign, the destructive process by which it is formed undergoing spontaneous healing by fibrous repair extending over many years. Very often, however, if no fracture has been sustained, a latent bone cyst remains, for despite a vigorous fibrous reaction the walls do not collapse. A fracture provides the necessary crushing and accelerates and completes the repair. Operation is therefore unnecessary. The injury is treated as if it were a simple fracture, and radiographs at intervals usually show progressive obliteration of the cystic cavity (Fig. 429).

Variants of bone cysts—If successive radiographs show failure of obliteration or progression of the bone destruction, the cyst should be explored and curetted. Any degree of fibrous osteitis may occur from the simple cyst with fibrous wall, or the almost solid fibrous mass with small cysts, to the solid fibromatous mass, with in many cases islands of giant cell tumour (Figs. 430-431). To ensure obliteration of the cavity and protection from recurrent fracture a bone graft may be employed.

Von Recklinghausen's multiple osteitis fibrosa^{3,4}—When cysts occur in many bones hyperparathyroidism must be suspected. There are multiple cysts bending deformities, general skeletal rarefaction and fractures through the cystic areas (Figs. 432-434). The serum calcium is raised from the normal 9.11 mg per cent to 12.20 mg. There is a marked increase in the urinary calcium output often with multiple renal calculi. The essential treatment is exploration of the parathyroid glands, removal of the adenoma, and a diet rich in calcium and vitamin D. If skeletal recalcification is still imperfect, the phosphorus intake must be controlled by the oral administration of alum acetate,⁵ because excess of phosphates in the blood provides a continued physiological stimulus to the glands and maintains a physiological hyperparathyroidism.

¹ Helfet. A New Conception of Parathyroid Function. *Brit Jour Surg*, 1949, xxvii, 651.
² E. J. Simha. Fibrocystic Diseases of the Bone. *Brit Jour Surg*, 1914, ii, 1.
³ J. Mandl. *Fernabst. f. Ch.* 1906, 11, 2.
⁴ D. Hunter. Calcium and Phosphorus Metabolism. *Lancet* 1929, vol. i, Nov. 14-19, 617.



FIG 428



FIG 429

Solitary benign bone cyst

Simple bone cyst in one of the common sites with pathological fracture. The forward bowing was corrected by manipulation. Three months later there is already evidence of obliteration of the cyst (Fig 429). Operation was unnecessary and the cyst was completely cured by the fracture.



FIG 430

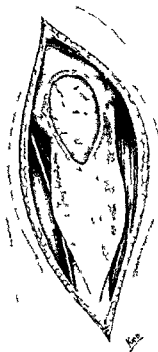


FIG 431

Solitary cyst—osteitis fibrosa

Pathological fracture through simple bone cyst (Fig 429). The cyst did not undergo spontaneous obliteration and was explored. It was of the solid fibrous type (Fig 431). After curetting a bone graft (shown in outline) was slid across the cavity.



FIG. 432



FIG. 433



FIG. 434

Hyperparathyroidism—osteitis fibrosa cystica

A parathyroid adenoma has caused generalised decalcification of all the bones, extensive cystic change (Fig. 433), bending deformity of the femur (Fig. 434) and pathological fracture of the tibia through a cystic area (Fig. 433). The fracture is surprisingly firm.

HYDATID DISEASE OF BONE

Hydatid disease¹ though rare in Great Britain and the United States still occurs with considerable frequency in the great sheep raising countries—Australia, South America and South Africa—despite measures to improve the conditions of slaughter houses and to prevent contact of domestic dogs with infected sheep. The greatest frequency has been in Iceland where no less than one seventh of the population were infected. In India and in China it is almost unknown. The fact that hydatid disease in man has almost disappeared in the British Isles despite the occurrence of the disease in the twenty five million sheep which are pastured is attributable to strict regulations of abattoirs, inspection of meat, good water supplies, the standard of public hygiene and care in feeding dogs. The *Tæma echinococcus* inhabits the small intestine of the dog. A single dog may harbour thousands of mature worms and pass many thousands of ova daily in the feces, thus contaminating food and water and infesting sheep or man—the intermediate hosts in which the larval stage of the worm develops with cyst formation. If dogs feed on the viscera of diseased sheep they become infested with scolices of which there may be many thousands in a single fertile cyst thus completing the life cycle of the tæma.

Clinical features.—Man is usually infested direct from the dog which he pets, caresses or allows to lick his hand, the ova being resistant and surviving in dust for many months despite desiccation, soaking, heating and freezing. Primary cysts develop in bone in about 1 per cent. of cases. They first appear near the epiphyses, usually in the femur or humerus and less often in the vertebra, tibia or pelvis. They grow so slowly over a latent period of many years and with such complete freedom from symptoms that the diagnosis is seldom established before adult life. Multiple irregularly shaped cysts closely resembling fibrocystic disease gradually develop without bone expansion but with steady infiltration along bony canals. Pressure, absorption of bone, invasion of the cortex and periosteum which offer little or no resistance and slowly progressive weakening lead finally and inexorably to spontaneous fracture. A fracture sustained without injury or with minimal injury is very often the first clinical manifestation of hydatid disease of bone. The fracture is painless because the nerves of the Haversian canals have been slowly and quietly destroyed. It is associated with little or no ecchymosis, swelling or cedema because the blood vessels have been destroyed and the bone is avascular. There is no crepitation because the bone has been destroyed. Non union is almost inevitable. Within a period varying from two to ten months after fracture as a result of the shedding of parasitic elements from bone into surrounding soft tissues a large cystic painless swelling develops. There is little or no inflammatory reaction and the clinical picture may resemble a cold abscess.

Differential diagnosis.—During the long period of latency early cyst formation discovered during routine radiography is often mistaken for fibrocystic disease. When spontaneous fracture occurs the bone destruction and total absence of periosteal reaction suggests secondary carcinoma. In the later stages of soft tissue dissemination cold abscess is simulated. The diagnosis may be confirmed by the Casoni and complement fixation tests.

and if necessary by diagnostic puncture. Microscopical examination of aspirated fluid shows fragments of laminated membrane, small daughter cysts and sometimes scolices or hooklets.

Treatment of pathological fractures—The prognosis is very grave because non union of the fracture is almost inevitable, extension rapidly occurs in soft tissues, neighbouring joints are involved and there is considerable risk of pre-operative or post-operative infection which may be fatal. No useful purpose is served by simple incision into the cysts, curettage or the application of formalin. These measures are almost invariably followed by recurrence. If the disease is still limited to bone and there is no invasion of soft tissues, total excision of the involved area followed by massive autogenous grafting is the treatment of choice. The excision must be sufficiently wide to include all cysts, however small. If soft tissues are already involved, amputation above the involved area is the only measure which offers the patient hope and it should be undertaken forthwith.

PAGET'S DISEASE—OSTEITIS DEFORMANS

Paget's disease¹ occurs most commonly in adult males, sometimes in a localised form when the tibia is usually involved but more often



FIG 435

Multiple cracks in Paget's disease



FIG 436

Sarcoma following Paget's disease

affecting many bones including the pelvis and skull. The bones are thickened and woolly and the cortex expands and encroaches on the medullary cavity. In the earlier stage of granular calcification fractures may occur. In the tibia multiple incomplete cracks may develop on the



FIG. 437



FIG. 438

Osteitis deformans—Paget's disease

Fig. 437—Case of pathological fracture of tibia, un united because it was inadequately immobilised.

Fig. 438—Case of pathological fracture neck of femur showing avascular necrosis of the head. The fracture united after five months' immobilisation. Nailing is contraindicated.

convex side of the bowed bone (Fig 43). If fracture occurs in a deformed bone advantage may be taken of the injury and the bowing should be corrected at the site of fracture. Repair is seldom slow, and if immobilisation is continued for several months firm union is secured. Non union is usually due to immobilisation for too short a period (Fig 437). Sarcoma sometimes occurs as a late development in Paget's disease¹ and if despite adequate immobilisation a fracture shows progressive decalcification and failure of union this complication must be considered (Fig 436). If operative reduction of the fractures of Paget's disease is needed internal fixation must not be contemplated. Even fractures of the femoral neck must be treated conservatively (Fig 438) for the bone is too fragile to withstand the pressure of nails or screws².

BENIGN BONE TUMOURS

Chondroma and chondromyxoma—Simple chondromas occur frequently in the phalanges and metacarpals and are often disclosed for the first time by a pathological fracture (Fig 439). The finger should be immobilised for a few weeks. The tumour is then explored, curetted and as a rule the cavity is filled with a bone graft (Figs 440-441). Cystic changes in the metacarpals and phalanges may also be due to osteitis fibrosa³ or to implantation dermoid cysts (Fig 442).

Osteoclastoma or benign giant cell tumour^{4,5}—The osteoclastoma occurs in adults usually in the lower end of the radius or femur or the upper end of the tibia. There is a pathological fracture in 14 per cent of cases. Radiographically the lesion is essentially destructive: there is bone expansion and the bony shell is extremely thin and often perforated. If fracture has occurred operative treatment is deferred for two or three months. The tumour is then excised. It is probably inadvisable to insert bone chips or a bone graft into the cavity for this appears to stimulate osteoclastic activity and to promote recurrence.

As an alternative to surgical treatment osteoclastoma can be treated successfully by radiation. The tumour gradually regresses and recalcifies and within a period of six to ten months final consolidation is secured. After successful treatment recurrence is exceptional. Although radiation treatment of osteoclastoma has been successfully developed in the United States relatively few cases have been so treated in Great Britain. Stanford Cade⁶ believes that this is due to the attitude of mind of the orthopedic surgeon to radiation and not the failure of radiation to cure osteoclastoma. He urges that radiation should be used as the first line of treatment and not as a measure to be adopted only when surgical intervention has failed.

Angioma of bone⁷—Hemangioma may occur in the spine⁸ and produce typical honeycombed rarefaction of the vertebral body with collapse and wedging which is to be distinguished from a simple crush fracture. The lesions usually heal under deep X-ray therapy. Angioma of the long bones gives

T. B. Davis "Supervention of Sarcoma in Paget's Disease" *Brit Jour Surg* 1933, xx, 699
 * Watson Jones "Pathological Fractures of Neck of Femur" *Brit Jour Surg* 1936, xx, 192
 * H. H. Platt "Cysts of the Long Bones of the Hand and Foot" *Brit Jour Surg* 1930, xv, 120
 J. Bloodgood "Conservative Treatment of Giant Cell Sarcoma" *Ann of Surg* 1916, 58, 217
 Gesseler and Copeland "Recurrent and Metastatic Giant Cell Tumors" *Arch of Surg* 1933, 20, 713
 * C. L. Little "Malignant Hemangioma of Bone" *Treatnt by Rad* 1921, 1, 121
 * Bucy, Paul and Capps "Hemangioma of Bone" *Amer Jour Roent and Rad Therapy* 1930, xii, 1
 * Ghorrini and Adson "Hemangioma of Vertebrae" *Jour Bone and Joint Surg* 1941, x, 25



Fig 439



Fig 440



Fig 441

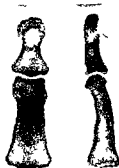


Fig 442

Simple cysts of phalanges

Fig 439—Chondroma with crack fracture Figs 440 441—Similar case cured and grafted
 Fig 442—Implantation dermoid cyst with fracture



Fig 443



Fig 444

Angioma of bone

Fig 443—Angioma involving the whole of the shaft of the humerus There is a characteristic soap-bubble appearance Complete destruction and pathological fracture of lower shaft

Fig 444—Photograph of patient showing large pulsatile swelling of limb The lady lived to a ripe old age and died of an intercurrent affection

a characteristic loose soap bubble appearance and long standing lesions may cause extensive destruction and pathological fracture (Figs 443-444)

MALIGNANT BONE TUMOURS

Osteogenic sarcoma—The osteolytic form of osteogenic sarcoma is an essentially destructive tumour which arises in the shafts of long bones most frequently in young adults and shows a central area of rapid bone destruction which dissolves the cortex without expanding it and very often causes pathological fracture. Early amputation offers the only chance of cure and is the best means of relieving the pain.

Ewing's sarcoma¹ is a rapidly growing tumour of early life. Pain is an outstanding feature and there is usually a rise of temperature even to 103° or 104°. The tumour is usually in the mid shaft of a long bone there is increased density, periosteal proliferation, expansion of the bone and mottling of the marrow cavity. It is not primarily destructive and pathological fracture is rare. The tumour shrinks under the influence of radiotherapy but the prognosis is poor and amputation is often necessary to prevent recurrence.

Multiple myeloma²—This is a rare tumour developing in many foci in the bone marrow of adults most commonly over fifty years of age. There is severe pain, tumour formation and in two cases out of three multiple pathological fractures. The fractures occur not only in the long bones of the extremities but very often in the ribs and sternum. The diagnosis is based on the multiple clear punched out lesions of the skull and other bones (Figs 448-449) and on the frequency of Bence-Jones albumoses in the urine. Union of fractures is slow but may be accelerated by radiotherapy. The disease pursues a fatal course usually within two years. It is seldom that a myeloma occurs as a solitary tumour (Fig 451).³ If there are no typical urinary or blood changes the tumour can be distinguished from an osteolytic sarcoma only by microscopic examination which shows the typical plasma cell.

Secondary carcinoma—*Carcinoma of the breast* is one of the tumours most frequently associated with bone metastases. The bones usually involved are the spine, pelvis, femur, skull, ribs and humerus in the order given. As a rule the lesions are multiple and most commonly they are osteolytic. There is no periosteal reaction, the primary focus is in the medulla with secondary invasion of the cortex and there may be diffuse mottling within the area of destruction (Fig 445). This mottling may differentiate multiple secondary carcinoma with involvement of the skull from multiple myeloma where the lesions are more clearly punched out.⁴ Pathological fracture occurs in 15 per cent of cases. The limb should be immobilised in the ordinary way for this is the best method of controlling pain and not infrequently the fracture unites and function is restored. *Carcinoma of the thyroid* may cause bone metastases with pathological fracture.

¹ J. Ewing, A Review and Classification of Bone Sarcoma. *Arch of Surg* 1921 1 42.

² J. Ewing, Neoplastic Diseases. Philadelphia W. B. Saunders Co. 1928 31.

³ Geschlatter and Copeland, Multiple Myeloma. *Arch of Surg* 1921 1 180.

⁴ Chester, A Solitary Plasmacytoma of Long Bones with Pathological Fracture. (Review of reported cases). *Lancet* 1935 111.

⁵ Bloodgood, Bone Tumours. Benign and Malignant. *Am J Surg* 1929 111 229.

⁶ Coslin, Pseudomyeloma and Carcinoma. *J of Surg* 1925 111 110.

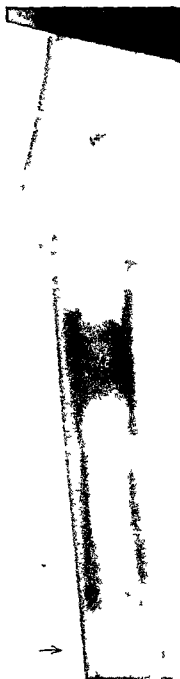


FIG 445



FIG 446



FIG 447

Secondary carcinoma

Fig 445—Metastases secondary to carcinoma of breast with pathological fracture. Note the second deposit in the lower shaft.

Fig 446—Secondary hypernephroma with pathological fracture of humerus. There is a second deposit in the scapula.

Fig 447—Secondary carcinoma of vertebrae showing bone destruction but minimal collapse because the bone is replaced by the tumour.



FIG. 448



FIG. 449

Multiple myeloma

Multiple tumours showing typical puncher defects of the skull (Fig. 448) and of the long bones where pathological fractures are sustained (Figs 450-451). The fractures unite slowly. Fig. 449 is a radiograph of the post mortem specimen and shows the tumour associated with one of the skull defects.



FIG 450



FIG 451

Pathological fractures of humerus and femur in multiple myeloma (same case as Figs 448 449)



FIG 452

Solitary myeloma

Rare case of solitary myeloma (plasmocytoma) of the upper shaft of femur with pathological fracture
Bony destroyed typical plasma cells

Carcinoma of the prostate usually produces slowly growing osteoclastic secondaries in the pelvis and spine. The invasive power of the growth is so moderate that bone proliferation can keep pace with it; the bone undergoes dense sclerosis and pathological fracture is rare.

Hypernephroma (renal carcinoma).—The metastases of hypernephroma have a predilection for bone; the lesion is usually destructive and pathological fracture occurs (Fig 446). The metastasis often occurs as a single focus and ten year cures have been reported after excision of the involved bone.¹ On the other hand metastases in bone may first appear many years after removal of the kidney, the interval being as long as ten years in some reported cases.² It is clear therefore that apparent cures must be accepted with reserve.

Neoplasms of the hæmopoietic system.—The lymphoid tissue marrow and phagocytic elements known as the reticulo endothelium are closely related and disease of these organs of a neoplastic type has a tendency to produce osteolytic bone lesions and pathological fracture. In Hodgkin's disease granulomatous deposits in the spine may be responsible for collapse of a vertebral body and lesions in the long bones sometimes cause pathological fracture. The chloroma, a greenish tumour related to the leucæmias is deposited in the skull and long bones of children approaching puberty and may be responsible for fracture. A fatal termination within six months is usual. In Hand Schüller Christian's disease an error of lipid metabolism accounts for extensive lipid deposits with destructive changes in the skull and bones of the extremities. The lesions are radio sensitive and re ossify under treatment. Gaucher's splenomegaly is another xanthomatosis of congenital origin in which a storage of lipid keratin is observed in lymphoid tissue and in long bones where pathological fracture occurs.

CONGENITAL FRAGILITY OF BONE

Osteogenesis imperfecta (*Fragilitas osseum*, osteopsathvrosis).³—In congenital fragility of bone there is a strong hereditary factor, some members of each succeeding generation suffering multiple fractures from the most trivial injury. The bones show few and widely separated trabeculae with an extremely thin cortex. After many fractures have been sustained the thin cortex becomes obscured by secondary thickening and sclerosis (Figs 444, 446). Three clinical types may be differentiated. In the *fatal type* the child is usually stillborn, the cranium is imperfectly ossified and there may be hundreds of fractures, some already united (Fig 447). The *infantile form* is less severe. There is stunting of growth and multiple fractures are sustained but the patient often survives. In the *adolescent type* the child appears normal at birth but there is the same susceptibility to fractures from trivial injury. In most cases blueness of the sclerotics is a striking feature (Figs 453, 454) and there is often otosclerosis causing deafness.⁴ Blueness of the sclerotics and otosclerosis may be present without obvious bone fragility in some members of an affected family. The fractures unite firmly and rapidly and they should always be treated in the ordinary way.

¹ Albrecht, *Arch f Klin Chir* 1905, 6, 10, 3.

² Broder, "Secondary Hypernephroma in femur with spontaneous fracture," *Phil Jour Surg* 1904, xl, 84.

³ E. A. Shank, *Br J Jour Surg* 1917, xv, 120.

⁴ Fraser, *Phil Jour Surg* 1914, xxi, 911.

⁵ M. S. Fox and S. J. Sweet, *Phil J Bones with Defects*, *Arch Otolaryng* 1910, xxxii, 40.



FIG 453



FIG 454

Fragilitas osseum (Osteogenesis imperfecta)

Colour photographs of mother and daughter showing typical blue sclerotics. Members of every generation of this family suffered multiple fractures from congenital bone fragility.



FIG 455



FIG 456

Fragilitas osseum

These are the radiographs of the femur and tibia of the sister of the patient shown in Fig. 453. She sustained twenty eight fractures in twenty five years.

After having sustained ten or twenty fractures in childhood the tendency may appear to die out and the patient subsequently leads a normal life.

Generalised osteopetrosis or Albers-Schonberg's disease (Marble bones, osteosclerosis fragilis)^{1,3}—This rare disease shows a familial tendency and is sometimes hereditary. It is of congenital origin and is characterised by extreme density of all the bones of the skeleton. The bones may resemble marble and show no tendency to fracture, but in other cases the hypercalcification is associated with brittleness and fragility and the resemblance

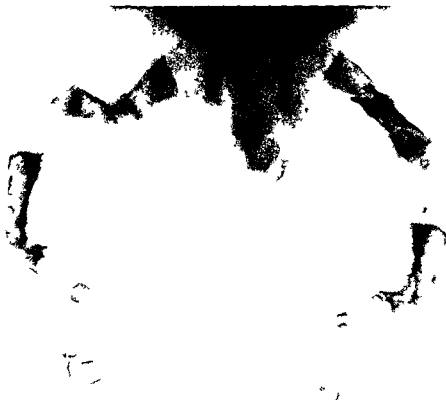


FIG. 457

Osteogenesis imperfecta

Ti fetal type of fragilitas osseum. The child was stillborn with multiple fractures of all long bones (radiograph of specimen in pathological museum, Liverpool University).

is nearer chalk than marble. The formation of dense bone usually continues until growth ceases, but very frequently there are intermissions, so that concentric rings of dense bone alternating with normal bone parallel to the epiphyses of the iliac crests may be a characteristic feature of the radiographs (Figs 458-462). The vertebral bodies may be uniformly dense, or there are dense layers of bone above and below, with a less dense layer between. The carpal and tarsal bones often show a circular distribution of dense bone surrounding a clear centre, or a dense centre surrounded by clearer bone. The phalanges may not be involved, but in some mild cases a dense

¹ Sear, Albers-Schonberg's Disease. *Brit Jour Surg* 1936 xiv 6.
² McClure and Braden, Osteopetrosis. *Amer Jour Dis Child* 193 x 265.
³ Fairbank, Increased Density of Bone. *Histol of the Marrow*. *Brit Jour Surg* 1939 xx 11.



FIG 458



FIG 459

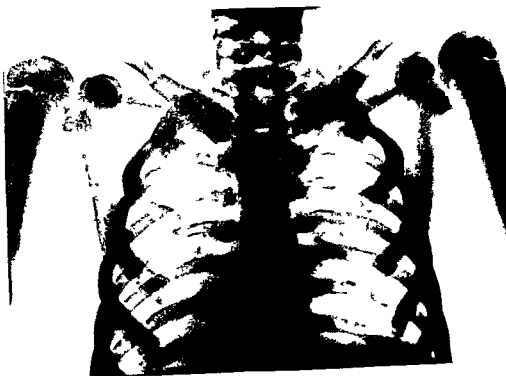


FIG 460

Albers-Schonberg's disease

The bones are hypercalcified and therefore fragile. Dense bone is laid down in concentric rings parallel with epiphyses (Fig 461). The skull is greatly thickened (Fig 458). Foraminal constriction causes cranial nerve palsies in this case of the left facial and auditory nerves (Fig 459). Knock knee is common. Fractures unite rapidly (Figs 461-462).



FIG 461



FIG 462

Albers-Schonberg's disease

band in the middle of the metaphysis is a striking feature. The skull is thickened, and foraminal constriction causes optic atrophy and sometimes other cranial nerve palsies such as facial paralysis (Fig 438-439). There may be calcinosis of the dura and of the kidneys vessels or ligaments. Anæmia is an almost constant feature of the syndrome and it is sometimes severe and fatal. Patients often die at an early age from intercurrent affections.

A somewhat similar generalised osteopetrosis has been described as an acquired disorder resulting from excess of fluorine in drinking water¹. In Madras India fluorine osteopetrosis has been endemic. Unlike true congenital osteopetrosis which is sometimes associated with bone fragility fluorine hypercalcification does not seem to predispose to fracture.

Cleido-cranial dysostosis²—This is not a disease of bone fragility but is congenital abnormality of ossification of the membrane bones in which one or both clavicles may develop in two halves. The history easily differentiates the condition from an united fracture of the clavicle, but in one case a victim made capital out of his disorder by concealing the history. He made a practice of tripping over the rope by which the brewer's drayman lowers barrels of beer into cellars. He then alleged that he had fractured his collar bone subsequently claimed damages and on three separate occasions was compensated for an un united fracture³.

¹ Fairbank. Increased and Decreased Density of Bone Fibres of the Marrow. *J. t. Jour. Surg.* 1939 xxvii, 1.
² J. Wilk. Two Cases of Fluorine Osteosclerosis. *Brit Jour. R. t. l.* 1941 xii, 213.
³ Fairbank. General Diseases of the Skeleton. *Brit Jour. Surg.* 19 xv, 120.

CHAPTER XII

BIRTH FRACTURES

Three types of fracture may occur in the newly born infant (1) fracture or epiphyseal displacement due to injury sustained during a difficult delivery, (2) multiple fractures associated with congenital fragility of bone, (3) congenital fracture of the tibia leading to congenital pseudarthrosis.

*Fractures due to birth injury*¹—The shafts of the long bones of the arm and thigh are involved more commonly than the epiphyses. The injuries in the order of frequency are (1) fracture of the shaft of the humerus, (2) fracture of the shaft of one or both clavicles, (3) fracture of the shaft of the femur, (4) depressed fracture of the skull, (5) displacement of the epiphyses of the humerus or femur. The fractures are seldom of the greenstick type. They are usually complete, and there is often considerable displacement. Large masses of callus develop, and the injury may be recognised only by the lump which becomes obvious two or three weeks after birth. Union is rapid, non union is practically unknown, and even if the fragments unite with angulation, alignment is restored by gradual moulding during the first few years of life. Shortening on the other hand may persist into adult life.

Fractures due to congenital fragility of bone—One or more fractures of long bones present at birth may be due to osteogenesis imperfecta. This has been discussed in Chapter XI (p. 278).

Congenital pseudarthrosis of the tibia—The solution of continuity of the tibia which may be present at birth is characterised by a curious indolence of the bones, and by a resistance to every form of treatment. It is a pathological entity of a very different type from the simple fractures of birth injury.

FRACTURE SHAFT OF HUMERUS

Clinical features—Fractures of the middle third of the shaft of the humerus almost invariably occur in breech deliveries during attempts to deliver the extended arms. The injury may also be produced by efforts to deliver impacted shoulders by axillary traction in vertex presentations. The fracture is transverse or spiral, overriding is rare, but there is usually outward angulation due to the pull of the deltoid muscle on the proximal fragment. Musculo spiral palsy is common, it usually recovers within six or eight weeks. There is stripping of periosteum leading to considerable callus formation and union is firm in about three weeks.

Diagnosis—Dangling of an arm by the side, or failure to move the limb

¹ Truesdell Birth Fractures and Epiphyseal Dislocations New York 1917

band in the middle of the metaphysis is a striking feature. The skull is thickened and foramenal constriction causes optic atrophy and sometimes other cranial nerve palsies such as facial paralysis (Fig. 458, 459). There may be calcinosis of the dura and of the kidneys, vessels or ligaments. Anæmia is an almost constant feature of the syndrome and it is sometimes severe and fatal. Patients often die at an early age from intercurrent affections.

A somewhat similar generalised osteopetrosis has been described as an acquired disorder resulting from excess of fluorine in drinking water.¹ In Madras, India, fluorine osteopetrosis has been endemic. Unlike true congenital osteopetrosis which is sometimes associated with bone fragility, fluorine hypercalcification does not seem to predispose to fracture.

Cleido-cranial dysostosis²—This is not a disease of bone fragility but is congenital abnormality of ossification of the membrane bones in which one or both clavicles may develop in two halves. The history easily differentiates the condition from an united fracture of the clavicle but in one case a victim made capital out of his disorder by concealing the history. He made a practice of tripping over the rope by which the brewer's drayman lowers barrels of beer into cellars. He then alleged that he had fractured his collar bone subsequently claimed damages and on three separate occasions was compensated for an united fracture.³

¹ Fairbank, Increased and Decreased Density of Bone & Losses of the Marrow, *Int Jour* 4, p. 1939 xxvii 1.
² J. W. Hale, Two Cases of Fluorine Osteosclerosis, *Int Jour Pathol* 1940 xlii 211.
³ Fairbank, General Diseases of the Skeleton, *Int Jour Surg* 1902 xv 129.



FIG 463



FIG 464

Birth fracture shaft of femur showing the usual right angled flexion of the proximal fragment (Fig 463) Treatment by strapping the limb to the trunk over corrects the deformity and fails to correct the shortening (Fig 464)



FIG 465

Birth fracture shaft of femur treated by strapping to the trunk six years after injury Although the angulation is perfectly corrected by remoulding there is still 1 in. of shortening Note the downward tilt of the pelvis on the injured side.

normally, may be due to a fracture of the humerus or clavicle to Erb's palsy, or to congenital syphilitic periostitis. The diagnosis of fracture is easily established by clinical and radiographic examination.

Treatment—The arm is strapped to the chest over a pad of wool which is thick enough to maintain moderate abduction of the limb. The hand and fingers are left free. The fracture may unite with some outward angulation, but this will be corrected during the first two to three years of subsequent growth. Perfect remodelling of the bone is possible even if there is 40° or 50° of angulation.

FRACTURE SHAFT OF CLAVICLE

Fracture of the clavicle occurs in the middle of the shaft and is nearly always due to direct digital pressure on the bone during traction on the shoulders to deliver the after coming head. There may be displacement of the fragments similar to that which occurs in adult fractures. The injury often escapes notice for two or three weeks until the lump due to callus formation becomes obvious. Both clavicles may be fractured simultaneously. A small pad of gauze dusted with talcum powder is placed in the axilla and between the arm and chest. a sling is worn for two weeks. No other treatment is necessary.

FRACTURE SHAFT OF FEMUR

Clinical features—Fracture of the upper shaft of the femur may be produced by torsion on the presenting leg in breech deliveries but is usually due to efforts to deliver a breech with extended legs either by groin traction or by bringing down the leg before delivery of the baby. The injury may also arise during extraction of the child by Caesarean section.

The normal attitude of the new born child is one of general and strong flexion, the hip joints are flexed to the right angle by tonic contraction of the psoas muscles. The proximal fragment of a fractured shaft of the femur is therefore strongly flexed and it usually lies at right angles to the distal fragment (Fig. 463).

Treatment—If such a fracture is treated with the thigh extended in a Thomas splint¹ in a plaster spica or in wooden spatula splints the fragments will unite with 90° of forward angulation. It is true that even this degree of deformity may disappear by the time the child is six or eight years of age but the shortening due to overlap and angulation does not disappear (Fig. 465). Crede suggested that the limb should be bandaged to the front of the infant's body with the hip joint almost fully flexed. This routine is often used but the deformity is then over corrected there is backward angulation and there may still be shortening (Fig. 464). The most rational treatment is to hold the limb at right angles to the trunk so that it lies exactly in the axis in which the proximal fragment is held by the psoas muscle. Overhead suspension of both lower limbs maintains the position and preserves full length. Several splints have been devised which achieve these objects satisfactorily.^{2,3,4}

¹ Robert Jones, "Treatment of Fracture of the Femur in the Newly Born," *Brit. Med. Jour.* 1904, i, 1254.
² Silver, "Treatment by Suspension of Fracture of Femur in Young Children," *Ann. of Surg.* 1909, xlix, 13.
³ Robinson, "Treatment of Birth Fractures of Femur," *Jour. Bone and Joint Surg.* 1934, xx, 78.
⁴ Moreno, "Obstetrical Fractures of Humerus and Femur," *La Prensa med. Argent.* no. 16, 14 Feb. 1935.

The depressed area of bone often undergoes spontaneous elevation, but if it fails to do so it should be elevated by operation. Uncorrected depressions and furrows may persist into adult life. The bone is easily sprung into position by a hook introduced through a small trephine hole at the bottom of the depression.

EPIPHYSEAL DISPLACEMENTS

Birth injury may cause displacement of the epiphyses at the lower end of the femur, lower end of the humerus or upper end of the humerus^{1 2}. These injuries are rare.

Lower femoral epiphysis—The centre of ossification is present at birth and the radiographic diagnosis of backward displacement presents no difficulty. The periosteum of the back of the lower shaft of the femur is stripped and displaced backwards with the epiphysis. If the displacement is not corrected the subperiosteal hæmatoma ossifies and a new shaft is formed behind the original one. The lower end of the original shaft is gradually absorbed. The radiographic appearances become quite normal in two or three years even despite complete failure of reduction. If any attempt is made to correct the displacement, care must be taken not to compress the popliteal vessels and so endanger the circulation of the foot. Traction must be applied to the limb before the knee is extended. The knee is immobilised for three weeks on a spatula splint or in a birth-fracture frame (Fig 466).

Lower humeral epiphysis—The centre of ossification of the lower humeral epiphysis does not appear until the latter half of the first year. If the epiphysis is displaced backwards by birth injury, radiographic diagnosis is impossible until ten or fourteen days after birth. Ossification is then seen beneath the displaced periosteum behind and to one side of the lower shaft of the bone. The clinical diagnosis may be made shortly after birth by the signs of helplessness and immobility of the limb, pain on attempted movement and swelling of the elbow. Traction is applied to the limb, and while it is maintained the elbow is gently flexed to just above the right angle. It is then supported in a sling. Even if the displacement remains imperfectly corrected, the regenerative powers of the infant are such that an apparently normal bone is reconstructed, exactly as in injuries of the lower femoral epiphysis.

CONGENITAL PSEUDARTHROSIS OF THE TIBIA

Whereas birth fractures of the femur, humerus and clavicle unite rapidly and with massive callus formation, congenital pseudarthrosis of the tibia is characterised by indolence of the fragments, minimal callus formation and non union which often persists despite prolonged immobilisation and bone grafting operations. One child was treated by continuous and uninterrupted immobilisation in plaster for four years without success³, another was operated upon fifteen times in a determined attempt to secure union, Putti reported eleven failures in thirteen bone grafting operations. Henderson⁴ recorded seven failures in eleven cases; amputation was often performed

¹ Michel. "Obstetrical Dislocation of Upper Humeral Epiphysis." *Rev. d'Orthop.* 1937, xxiv, 201.

² Seagilletti. "The Obstetrical Shoulder Trauma." *Surg. Gyn. Obst.* 1934 lxxi, 663.

³ B. L. Macfarland. "Birth Fracture of the Tibia." *Brit. Jour. Surg.* 1940 xxviii, 707.

⁴ M. L. Henderson and R. S. Clagor. *Proc. Mayo Clinic* 1941 xxi, 769.

The birth fracture frame which has been made for me is of the simplest possible type and has proved effective (Fig 466) Bandage is firmly bound over the base and on this the infant lies there are two notches on the crossbar and extension strappings from each lower limb are fastened to them The limbs must be held with sufficient tension to raise the buttocks from the bandage slings Traction on the limb may be increased by bandaging the trunk of the infant to the frame In this frame nursing presents no problems, napkins are used in the ordinary way and there is no difficulty from pressure sores Union is usually quite firm in four weeks and the apparatus is then discarded

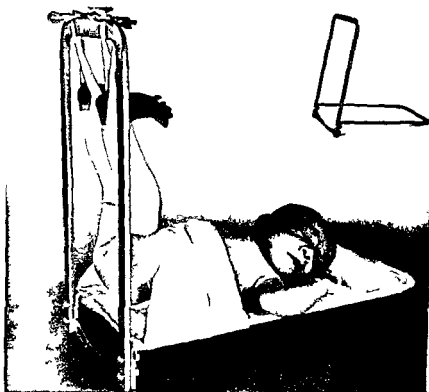


FIG 466

Birth fracture frame for injuries of the femoral shaft The overhead suspension maintains length and keeps the limb in the direction in which the proximal fragment is held by the psoas muscle

DEPRESSED FRACTURE OF SKULL

The pliability of the skull and the wide open sutures protect the cranial bones of the infant from fracture Even severe intracranial birth injuries accompanied by asphyxia circulatory and respiratory failure and cerebral hemorrhage with spastic diplegia are usually unaccompanied by fracture If a fracture is sustained the parietal bones are usually involved and the injury is due to pressure of the head on the bones of the pelvic inlet particularly the promontory of the sacrum in delivery through a slightly contracted pelvis Attention may be drawn to the injury by the overlying subperiosteal hematoma

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¹ Miel et al. "Obstetrical Dislocation of Upper Humeral Epiphysis." *Rev. d'Orthop.* 1911, xxiv, 201.

² Scaglietti. "The Obstetrical Shoulder Trauma." *Sur. Gyn. Obst.*, 1938, lxxv, 869.

³ B. L. Macfarlane. "Birth Fracture of the Tibia." *Brit. Jour. Hyg.* 1940, xxxvii, 8.

⁴ M. S. Henderson and R. S. Clegg. *Proc. Mayo Clin.* 1941, xvi, 60.

after many years of treatment because limbs were shortened atrophic and functionally useless

The pathology is not yet fully understood. No abnormality has been found in the blood calcium phosphorus or phosphatase levels there is no hereditary factor and the condition is seldom associated with congenital deformities or developmental errors. Patches of café au lait pigmentation of the skin have been reported in some cases but not in all¹. The proof that pseudarthrosis of the tibia is not a true congenital abnormality lies in the fact that the child is often normal at birth. Three types may be recognised: (1) the infant is born with a fracture of the tibia which was sustained before or during birth and which fails to unite. (2) the infant is born with angular deformity of the tibia but no fracture and pseudarthrosis develops in later months or years as the result of fracture or osteotomy^{3,4}. (3) the infant is normal at birth but sustains a fracture of the tibia with pseudarthrosis in early childhood. It is clear therefore that congenital pseudarthrosis and birth fracture are misleading terms. I personally doubt whether any congenital or constitutional abnormality exists. I think it is more likely that pseudarthrosis is due to the same local conditions which cause non union of tibial fractures in adults. This is suggested by three features which are common to all cases of pseudarthrosis in infants: (1) the site of fracture (2) the angulation of the fragments (3) the instability of the fracture and difficulty of immobilisation. The site of fracture near the junction of the middle and lower thirds of the shaft of the tibia is a level where the blood supply of the bone is feeble and where fractures in adults often show marked indolence minimal callus formation and slow union in striking contrast with shaft fractures elsewhere. The delaying influence of a poor blood supply was discussed in Chapters I and II and it was shown that slow union from this cause often leads to non union if there is also shearing strain and imperfect immobilisation. One important source of shearing strain which only recently has gained full recognition is weight bearing on an angulated bone (Chapter XXV). This obviously applies to tibial fractures in infants because a marked degree of angulation is almost invariable in congenital pseudarthrosis and early weight bearing has long been encouraged in the hope of stimulating union. Moreover the factor of imperfect immobilisation known to be so important in adult fractures is still more evident in the fractures of infants. Even when there is no fracture it is most difficult to immobilise the elastic bones and joints of infants. Every orthopaedic surgeon is aware of the incredible facility with which a babe can wriggle his limb out of a plaster applied from toes to groin with right angled flexion of the ankle and knee joints. How much more readily can a child wriggle his limb when there is also a fracture especially when it is a fracture with complete lack of natural stability owing to the conical shape of the pointed fragments and the intervening gap. In the infant no matter what care is used it is impossible to immobilise a fracture of the tibia completely by the external fixation of splints or plaster. Even the internal fixation of a bone graft is inadequate if a single autogenous graft is used because the bone is so slender and fixation so difficult. If it is true that the union of

B. H. Moore Orthopaedic Relationship of Neuroblastomatous Jour. Bone and Joint Surg. 1941 xx:1 109
 * M. Canavali Le pseudarthrosi congenite del tibiale. Chir. d'org. e. movim. n. 1930 x 1 18
 E. Caspe Malformations asymétriques des extrémités. J. de l'orthop. 1907 viii 24
 H. Hallock Pseudarthrosis following fracture of the tibia. Jour. Bone and Joint Surg. 1938 xx 614

fractures of the shaft of the tibia in infants is dominated by the same influence of blood supply as the union of fractures in adults and that the same complete and prolonged immobilisation is needed, the source of congenital pseudarthrosis is explained. This would be confirmed if a new method of grafting was devised and it was found that complete internal fixation and complete protection from angulatory strain was always successful in securing union. Recent experience¹ suggests that this may now have been accomplished and supports the view that failures in the past were due not to obscure pathological changes in the bone but to imperfect operative technique and reliance on slender grafts inadequately fixed to angulating bones.²

The first point in the technique is that an inlay graft can seldom hope to succeed because it is necessarily more slender than the very slender host bone; moreover the preparation of a bed for an inlay graft involves excision of the conical ends of the fragments and serious increase in the gap between them. An onlay graft must be used so that all host bone is preserved (Figs 467-468). In the second place the fixation of a single graft to atrophic attenuated fragments presents almost insuperable difficulty and it is doubtful whether such grafts were ever completely successful in controlling angulatory strain. Double onlay grafts must be used one on each side of the pseudarthrosis fixed securely to each other with the host bone clamped between them as if in a vice. Sufficient strong bone for dual grafts can seldom be secured from the skeleton of the infant and maternal grafts are usually necessary. Finally the fixation of the grafts, the prevention of recurrent angulation and the control of strain at the site of pseudarthrosis must no longer depend on the devices formerly used—fragments of wire and pieces of catgut which any carpenter would laugh to scorn. Screws which completely fix the grafts and transfix the host bone must be used. Recent metallurgical research with stainless steel, vitallium and other alloys has made it possible to use screws without fear of reaction and loosening in the bone, and it is this development in surgical equipment which has made the modern technique of onlay grafting possible and provided the key to the problem of congenital pseudarthrosis of the tibia (Figs 469-471).

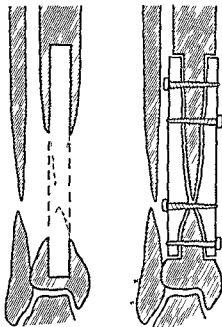


FIG 467

FIG 468

In congenital pseudarthrosis of the tibia an inlay graft is difficult to fix and it seriously increases the gap between the fragments (Fig 467). These difficulties are met by double onlay grafts screwed to each other (Fig 468).

Before the introduction of vitallium screw fixation for double onlay grafts,

H. B. Boyd, Congenital Pseudarthrosis and Treatment by Dual Bone Grafts. *Jour. Bone and Joint Surg.* 1941, xliii, 497.

¹ Henderson, in reporting four successes in eleven operations, records that even in the successful cases the grafts broke and the patients seemed doomed to go on with non union, but slowly under supportive measures and the use of braces over a long period union developed. In the other seven cases failure was complete. Could more honest acknowledgment be made of the failure of operative technique in forty years? Pseudarthrosis of the Tibia. *Proc. Mayo Clinic* 1941, xvi, 69.



FIG. 469

Congenital pseudarthrosis of the tibia



FIG. 470



FIG. 471

An attempt to bridge the pseudarthrosis by transposing the fibula failed. Double onlay maternal grafts were used (Fig. 470). After immobilisation in plaster for nine months union was soundly consolidated (Fig. 471).

Macfarland¹ recorded successful results from a by pass technique of bone grafting (Fig 472). Without attempting to correct the deformity, a massive

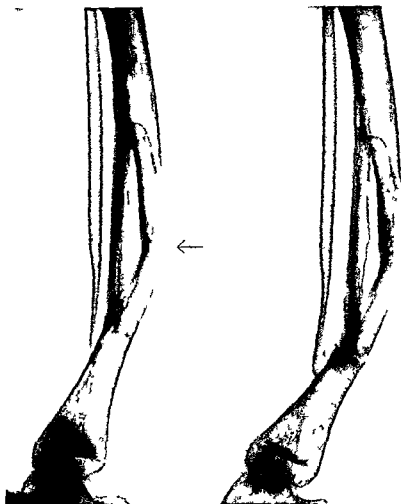


FIG 472

FIG 473

A congenital pseudarthrosis of the middle third of the tibia was treated by a bridge graft and it united (Fig 472) but at the lower end of the graft where there was still angulatory strain due to the forward bow of the tibia a second pseudarthrosis developed (Fig 473) presumably at the site of a fine crack sustained during the grafting operation.

graft was wedged on the concave side of the angulated bone at a distance from the pseudarthrosis which was bridged by the graft. He believed that one reason for his success might be the lack of direct contact between graft and pseudarthrosis where it appeared that osteolysis was almost malignant in its capacity to absorb all bone within reach. The true importance of his technique was the secure fixation of a stout graft to a broad base of bone well above and well below the site of pseudarthrosis in such a position that the inhibitory effect of angulatory strain was excluded by short circuiting the angulation. This is confirmed by an interesting case in which I performed

¹ B. L. Macfarland: Birth Fracture of the Tibia. *Br. J. Surg.* 1940 xxxii 104. (This article also records success from double onlay grafting but vitality is secure fixation as not then employed.)

Macfarland's operation The pseudarthrosis was bridged by a stout graft so that the line of weight bearing passed through the graft and no longer through the angulated bone The pseudarthrosis promptly united But the degree of angulation of the tibia was such that weight bearing still passed in front of the ankle joint there was still a forward bow of the tibia at the site of impaction of the lower end of the graft and at this level a second pseudarthrosis developed (Figs 472-473) It is presumed that a slight crack was sustained at the time of the grafting operation which was slow in healing so that after several months with the renewal of weight bearing and angulatory strain the crack widened to an obvious fracture and developed all the characteristics of congenital pseudarthrosis A further operation was performed alignment was corrected at the second pseudarthrosis so that weight was transmitted accurately through the tibia from knee to ankle and fixation was maintained by only grafts screwed to the host bone Further progress was uneventful

PART III
INJURIES OF THE TRUNK AND HEAD

CHAPTER XIII

FRACTURES OF TRANSVERSE AND SPINOUS PROCESSES

CLASSIFICATION OF VERTEBRAL INJURIES

Fractures of the spine may be divided into three groups corresponding to the three anatomical components of the vertebra—the muscular processes the vertebral body and the neural arch. The injuries which may be sustained are closely related to the function of these structures. (1) the transverse and spinous processes serve for the attachment of muscles they are usually fractured by muscular violence and displacement of fragments is always due to the retraction of muscles. (2) the vertebral bodies transmit weight they are fractured by the compressing force of excessive weight bearing and displacement is due to the superimposed weight. (3) the laminae articular processes and pedicles protect the spinal cord and nerves they are fractured by a shearing force and displacement is usually associated with injury to the nerves.

FRACTURES OF TRANSVERSE PROCESSES

Etiology—Injuries of the transverse processes are almost confined to the lumbar region. The quadratus lumborum arises from the crest of the ilium and is inserted into all five lumbar transverse processes and to the twelfth rib. A sudden unguarded contraction of the muscle may crack one or more processes. In other cases a fall from a height or the dropping of a heavy weight may force the trunk to the opposite side and in attempting to resist the movement the powerful contraction of the quadratus lumborum tears it away from its insertion (Fig. 474). Several transverse processes and sometimes all the processes on one side are avulsed. After severe crushes a line of injury may be traced from the pelvis through the transverse processes to the ribs which are also fractured.

Severity of the injury—The radiographic picture conveys a false impression of the injury. The small detached bone fragments appear trivial and unimportant. A short disability period is expected. When it is found that the disability is prolonged there is a temptation to blame the patient and accuse him of neurasthenia or even of malingering. If attention is paid to the soft tissues between the fragments the severe nature of the injury is more easily recognised. There is a widespread hematoma. Muscles, fasciae and aponeuroses are torn. Blood vessels are damaged. The sensory nerves traversing the region are stretched or avulsed and in the later stages they may be compressed in the scar tissue (Fig. 475). There is extensive traumatic exudation which leads to scar tissue and adhesion.

formation and the exudation is increased by early movement and passive stretching. It is certainly our duty to be optimistic and to encourage the patient. But to go to the lengths sometimes advised of refusing to immobilise the torn structures of reassuring the victim that his back is not broken or instituting immediate exercises and passive stretching and of urging an early return to work is wrong. The lacerated tissues must be



Fig. 474

Fractures of the third fourth and fifth lumbar transverse processes on the right side with subperiosteal ossification in the origin of the quadratus lumborum from the ilium. The first lumbar transverse processes are developed separately as lumbar ribs.

immobilised. Functional activity should be instituted at an early stage but mobilising exercises must be deferred until repair is sound.

Diagnosis—There is severe pain in the flank and tenderness on deep pressure in the interval between the spinal and abdominal muscles. Pain is increased by passive stretching of the trunk to the opposite side and by active flexion against resistance to the same side. Hyperextension of the hip by pulling on the psoas muscle also increases the pain.

Errors in the radiographic diagnosis may arise unless care is taken to distinguish the margins of gas shadows in the colon and of the psoas muscles which cross the shadows of the transverse processes. On one or both sides

of the first lumbar vertebra the transverse process may be congenitally separated to form a short lumbar rib (Fig 474). The epiphyseal lines of the transverse processes must also be distinguished.

Treatment—A crack through one or two transverse processes without separation of the fragments does not require immobilisation in plaster. The lumbar region is firmly strapped and bandaged for three weeks.

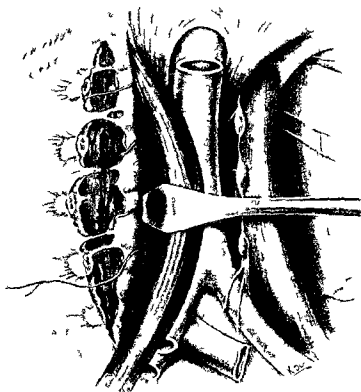


FIG 473

Fractures of the transverse processes are due to avulsion of the *quadratus lumborum* and the injury is more severe than the radiograph suggests. There is extensive tearing of fasciae, aponeuroses, muscles, blood vessels and nerves.

Cautious activity is resumed within a few days and full activity is encouraged after four weeks. In these cases recovery is rapid and the disability is unimportant.

If avulsion of several transverse processes shows that there has been extensive tearing of the lumbar muscles, a plaster jacket is applied over a single layer of stockinet extending from the lower pelvis to the nipple line and it is kept in position for six or eight weeks. Recumbency is unnecessary. Within a few days the patient should dress normally. After two or three weeks when the acute pain has subsided exercises are practised for the spinal musculature (pp 314-318). The protection of the plaster jacket is sufficient to prevent stretching of the repairing tissues.

After eight weeks the plaster is discarded. No posterior spinal support should be prescribed. Mobilising exercises are necessary, and they are gradually increased. The resumption of work and of recreational activity is usually possible within from three to six months.

The transverse processes may fail to unite by bone. This is not of any significance. The injury is essentially a muscle rupture rather than a fracture, and fibrous repair is to be expected.¹ Removal of detached fragments is not indicated. After the less severe injuries, localised adhesion formation may be relieved by manipulation of the spine performed some months after injury. On the other hand more severe and extensive ruptures cause such dense adhesion formation that manipulation is seldom of value. Contracture of the scar tissue especially when there has been no immobilisation, may even be sufficient to produce a well marked lumbar scoliosis.

FRACTURES OF SPINOUS PROCESSES

"Clay Shovellers' Fracture"

In the fully extended position of the trunk the spinous processes are protected from direct injury. In the flexed position, one of the long upper dorsal spinous processes may be fractured by the blow of a cricket ball or of a hammer, brick or other object dropped from a height. More often the spinous process, usually of the first dorsal or seventh cervical vertebra, is avulsed by muscular violence.² Under the title of "Shovellers Disease" Debuch³ reported 187 cases observed within a period of sixteen months and Matthes treated 107 cases in two years. In this country few surgeons have seen more than one or two isolated cases.⁴

The injury is sustained almost invariably by labourers shovelling dirt or clay. During the act of driving in the shovel, or during the upward thrust into the air, the patient experiences a snapping sensation between the shoulder blades followed by progressive pain which makes it impossible to continue work. Radiographic examination shows a fracture of the seventh cervical or first dorsal spinous process. In some cases the terminal half of the process is completely avulsed and displaced downwards by the pull of the rhomboid muscles. Immobilisation is unnecessary, and operative excision of the detached piece of bone as recommended in recent reports⁵ also seems to be unnecessary. Despite fibrous union of the fragment, patients usually return to work free from symptoms in about five weeks.

¹ R. K. Clonlough and Hoffmann "Fractures of Vertebral Processes" *Proc Mayo Clin*, 1940, xxii, 1.

² Zollinger "Isolated Spinous Process Fractures—Schlipper's Disease (seventy-eight cases)" *Archiv. Med. Wchnschr.* 1931, 48, 503.

³ Matthes Debuch *Arch f Orth und Unfall Chir.*, 1931, xxxvii, 2.

⁴ Watson Jones "War Injuries of the Spine" *Proc Roy Soc Med*, 1941, xxxiv, 464 (section on Surgery, 41).

⁵ McKellar Hall (report of 15 cases: 11 in men shovelling clay, 1 shovelling gravel, 1 shovelling lime and 1 pitting clay) *Jour Bone and Joint Surg*, 1941, xxii, 63.

CHAPTER XIV

FRACTURES OF DORSO-LUMBAR VERTEBRAE

Fractures of vertebral bodies are usually sustained at the junction of mobile and relatively immobile segments of the spine. The lumbo dorsal and cervico dorsal junctions are therefore involved and the most frequent injury is to the twelfth dorsal and first lumbar vertebrae. Most fractures are sustained while the spine is in flexion. extension fractures are extremely rare.

TYPES OF VERTEBRAL BODY FRACTURE

1. Flexion Fractures

The direction of the flexion force determines the type of fracture—(1) the patient falls from a height in the standing or sitting position, the spine is in slight flexion and vertical compression causes wedging of one or more vertebral bodies. (2) the patient is stooping and a heavy weight falling on the shoulders sharply angulates the spine into acute flexion, a comminuted fracture of one vertebral body results. (3) the patient is struck behind the shoulders by a moving object such as a falling weight



FIG. 476



FIG. 477



FIG. 478

The three varieties of flexion fracture of vertebral bodies—simple wedge fracture due to vertical compression (Fig. 476), comminuted fracture due to acute flexion angulation (Fig. 477), fracture dislocation due to flexion with traversing momentum (Fig. 478)

(Figures 46 to 502 have been reproduced by courtesy of the "Journal of Bone and Joint Surgery" from the author's articles in vol. xxi, 1931 and vol. xx, 1934.)

or a motor vehicle which flexes the spine and adds a traversing momentum, the forward displacement of the upper segment causes a fracture dislocation. The nature and extent of the injuries and the treatment and prognosis is different in each of these three types.

Simple wedge or compression fracture—This is the commonest group of spinal fractures and it includes 60 per cent of vertebral body injuries. The wedging is due to diffuse bending with vertical compression (Fig 479). As a rule more than one vertebra is involved. Two, three, four or even five bodies may show reduction of the anterior vertical depth. In some cases the wedging appears to be confined to one vertebra, but actual measurement shows slight diminution of the anterior depth of the bodies above and below.



FIG 479

Unreduced wedge fracture of vertebral body

The patient may have fallen from scaffolding, from a crane, from a ship's deck, from a ladder or from a roof. The result is the same whether the victim strikes the ground in the sitting or in the standing position. For example, one blade of the propeller of an aeroplane broke off in the air, the plane settled to the ground like a leaf, with the pilot and his wife still sitting in the cockpit, and both victims sustained wedge fractures of the lumbar vertebrae. A workman fell 50 ft and landed upright in soft soil. Although his back had not come into contact with any object, he had fractured his lumbar spine. A sailor fell from the crow's nest down the inside of a ship's mast and fractured both heels and the lumbar spine. The association of wedge fracture of one or more vertebral bodies with crush fracture of the os calcis is not uncommon, and when either of these injuries is found,

the patient must be specially examined to exclude the other.

In this type of injury the elastic intervertebral discs escape damage. There is no fracture of the pedicles or laminae, and forward tilting of the bodies is possible only through movement of the interarticular joints. These joints are not actually dislocated but they are usually strained.

Comminuted fracture—Ten to fifteen per cent of vertebral body injuries are comminuted fractures. The fracture is the result of a more localised angulation of the spine such as occurs from the fall of a weight on the back of the shoulders. The injury occurs in coal mines from falls of roof. There is such sharp angulation that the anterior angle of one vertebral body is driven into the middle of the adjacent body. This produces a triangular depression of the intervertebral surface which corresponds exactly in size and shape with the anterior angle of the vertebra above (Fig 480). Occasionally both upper and lower surfaces are similarly depressed, and anterior marginal fragments have been squeezed forwards.

It is important to recognise the comminuted type because union is much slower than after simple wedging and compression. Moreover the inter-

vertebral disc is often ruptured and narrowing or disappearance of the disc space may cause slight kyphosis despite complete reduction of the bone. The injury is so localised that one pair of interarticular joints take the whole strain and pain often persists unless early operative fusion of the spine is performed. In many cases however the disc is so damaged that bony ankylosis occurs between the vertebral bodies and pain is then relieved. In rare cases of comminuted fracture the impaction of the adjacent vertebra not only displaces anterior marginal fragments forwards but also displaces posterior marginal fragments backwards into the neural canal. There is then a considerable danger of spinal cord injury and special treatment is necessary (Chapter XV).

Fracture dislocation.—In about 20 per cent of dorso lumbar vertebral body fractures there is forward dislocation of the upper segment of the spine

(Fig 481). The injury is primarily a dislocation of the intervertebral joint and wedging or comminution of the vertebra below is incidental. The neural arch injury is a fracture of the pedicles or laminae or a dislocation of the interarticular joints. The spinal cord and nerves may be damaged by contusion or compression and the treatment may be dominated by the paraplegia. Nevertheless the bone displacement is comparable with simple compression and comminuted fractures in that it is due to flexion force and that it is corrected by extension.

In some cases of fracture dislocation with bilateral interarticular joint dislocation the upper segment of the spine is not only dislocated forwards but it is also displaced to one side in such a way that the articular processes are locked. These injuries are particularly dangerous and special treatment is necessary (see Chapter XV).



FIG 480

Unreduced comminuted fracture of vertebral body



FIG 481

Unreduced fracture-dislocation of vertebrae

2 Extension Fractures

The anterior common ligament is of such strength that rupture or avulsion of the ligament from its bony insertion by hyperextension strain is almost impossible. Hyperextension fractures are therefore extremely rare. They will be considered together with other exceptions to the usual

treatment, in Chapter XV. It is this strength of the anterior common ligament and of the anterior attachments of the intervertebral disc which makes it possible to reduce flexion fractures of the spine by hyperextension. Whether the fracture is a simple wedge compression, a comminuted fracture or a fracture dislocation, the displacement is corrected by the tension of the anterior common ligament which is produced by hyperextension of the spine.

CLINICAL AND RADIOGRAPHIC DIAGNOSIS¹

Clinical signs—The important clinical sign is prominence of one spinous process. Even if this is not visible the prominence can be felt when the examining finger is passed down the line of spinous processes. The patient usually but not always complains of pain, careful examination is necessary whether there is pain or not.

Radiographic diagnosis—Radiographic examination is essential. The lateral view discloses the injury more clearly than the antero-posterior view. Even trivial wedging of a vertebral body, or slight buckling of the upper or lower margins must be accepted as a complete fracture. The position shown in the radiograph does not represent the maximum deformity which is possible. Leverage of the spine above and below the injury is so powerful that any movement alters the deformity. In the extended position deformity is minimal, and yet as soon as flexion is permitted wedging appears. If the spine is not immobilised in full extension the bone will absorb the wedging increase, and severe deformity develop.

"Kummell's disease"—This condition was first described before lateral radiography of the spine was available.² It was believed that a vertebra though not actually fractured could be injured, and that delayed deformity supervened after many months or years. There is probably no such condition. Kummell's disease is simply an overlooked fracture of the spine in which failure to immobilise the injury has caused progressive wedging and absorption of bone.^{2,3}

Radiography in flexion—If routine radiographs are inconclusive, further films may be taken with the spine in flexion. This often discloses the injury more clearly (p. 159).

Repeated radiographs after an interval—If there is still doubt, the examination should be repeated ten to fourteen days later. The flexion movements permitted in the interval will bring to light any suspected fracture (p. 155).

Differential diagnosis—Wedging of the lower dorsal vertebrae in Scheuermann's disease is often mistaken for a crush fracture, especially when the disease has developed so gradually that there have been no symptoms and the patient has accepted the slightly rigid round back as normal. The disease develops after puberty, involves the seventh to the tenth dorsal vertebrae and accounts for a rounded low dorsal kyphosis. All the bodies are equally wedged and there is equal narrowing of all the intervertebral discs. There may be herniation of the nucleus pulposus into the adjacent bone causing small depressions of the intervertebral surfaces (Fig. 482). In later years there isipping of the anterior margins of the vertebral bodies (Fig. 483). These features and the rarity of crush

¹ Kummell, *Arch f Klin Chir* 1921 118 876

² Watson Jones, *Brit Med Jour*, 1932 1 397

³ Hosford, Kummell's Disease of the Spine, *Lancet* 1936 270 219



FIG 482

Scheuermann's disease in patient aged eighteen showing narrow discs, wedged bodies and hernial protrusions of nucleus pulposus



FIG 483

Old Scheuermann's disease in patient aged thirty five showing narrow discs, wedged bodies and lipping of the vertebral body margins

fracture in this particular region serve to differentiate the disease. Kyphosis due to congenital wedge vertebra and to the destructive changes of tuberculous disease and secondary neoplasms is more easily differentiated by the typical radiographic appearances.

PRINCIPLE OF REDUCTION BY HYPEREXTENSION

The fracture is produced by forcible hyperflexion. When the compact layer of the vertebral body is fractured the framework of cancellous bone collapses like a concertina. The pivot of collapse is the interarticular joint. Similarly the interarticular joint may be made the fulcrum for reopening the bone when the spine is once more extended. The annulus fibrosus of the intervertebral disc and the attachments of the anterior common ligament anchor the vertebrae together and simple hyperextension of the spine completes the reduction.

Davis' suspension technique—In 1929 Davis¹ advocated extension of the spine by overhead pulley traction on the feet of the prone patient who was fully anaesthetised (Fig 484). Manipulation was then made by several quick but measured downward thrusts at the sides of the kyphosis. A plaster bed was made, extending from the head to the knees and in this the patient lay for several months.

¹ A. G. Davis *Jour. Bone and Joint Surg.* 1929 xl 133 and 1934 xx 472

Watson-Jones' postural reduction^{1*}—In 1930 a method of postural reduction was described. The conscious patient lay prone between two tables so that the spine gently sagged to the normal limit of hyperextension (Fig 485). No manipulation, traction, or direct pressure was employed.

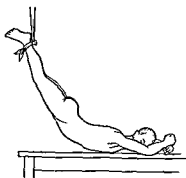


FIG 484

Davis suspension technique for reduction of vertebral body fracture

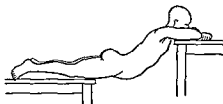


FIG 485

Watson Jones technique for postural reduction of vertebral body fractures

Anæsthesia was unnecessary. The patient's body weight was sufficient to reduce the fracture. A plaster jacket was applied from the groins to the clavicles and within a few days exercises and simple recreations were resumed.

Methods of controlled extension—Since that time many other methods of achieving extension of the spine have been recorded. The prone patient is supported on a ventral sling or hammock, or on the canvas of a Rogers' frame^{2,4}, or the supine patient is laid on a curved strip of metal, on a motor jack,⁵ or on the kidney bar of an operating table which is slowly pumped up (Fig 486). It might appear that all these procedures achieve the same

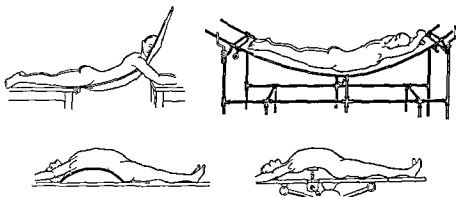


FIG 486

Reduction of vertebral body fractures by controlled degree of extension. The pelvis does not fully tilt forwards; the limit of hyperextension may not be reached and reduction is often imperfect.

frame^{2,4}, or the supine patient is laid on a curved strip of metal, on a motor jack,⁵ or on the kidney bar of an operating table which is slowly pumped up (Fig 486). It might appear that all these procedures achieve the same

¹ Brit. Orth. Assoc. Annual Meeting 1930. Report *Jour. Bone and Joint Surg.* 1931 xlii 283.

² Watson Jones, *Brit. Med. Jour.*, 1931 p. 304, and *Jour. Bone and Joint Surg.* 1931 xli 30.

³ Rogers, An Extension Frame for the Reduction of Vertebral Body Fractures. *Surg. Gyn. Obst.*, 1930 111.

⁴ Rogers, Treatment of Fractures of Vertebral Bodies. *Arch. of Surg.* 1931 xxx 284.

⁵ Johnson, Automobile Jack for Fractured Spine. *J. Amer. Med. Assoc.* 1931 xlii 56.

object but in fact they are essentially different from the original technique. Whereas by the first methods the unsupported trunk finds its own limit of hyperextension by these methods the surgeon determines the degree of extension and supports the trunk in that position. But it is not enough to extend the spine it must be hyperextended and it must be hyperextended to the limit of this movement. Only when this limit has been reached is the anterior ligament sufficiently taut to complete the reduction. The limit of hyperextension varies considerably in different patients and the surgeon cannot possibly estimate it the spine must be unsupported so that it finds its own limit. Moreover the pelvis must be fully tilted forwards and the abdominal muscles put on a full stretch. A ventral sling adds to the patient's comfort during reduction by preventing this tight stretched feeling but in so doing it prevents complete reduction. For the routine treatment of lumbar and lumbo dorsal fractures the two table postural method is the most simple. If for any reason the surgeon must use general anaesthesia the Davis suspension technique is better because the patient's co-operation is a necessary part of the postural method.

The method of postural reduction of fractures of the spine is so simple that there is a danger of overlooking details which appear trivial but which make the difference between success and failure. Incomplete reductions with imperfect results are often seen. The technique must be considered in detail.

TREATMENT OF DORSO LUMBAR FRACTURES—COMPRESSION TYPE

First aid treatment—Only the surgeon who has actually reduced vertebral fractures and has seen the kyphos smoothly disappear as the spine was extended can fully appreciate the power of the levers represented by the segments of the trunk above and below the injury. Gentle extension movement even in a conscious patient corrects the displacement similarly gentle flexion movement increases the displacement. If the victim is lying face upwards and is lifted in the usual way by the shoulders and legs the spine is strongly flexed and the cord is in danger of compression (Fig 487). Cases of suspected spinal injury must never be lifted in this way. Sagging between the points of support must be prevented. Roller towels or broad slings must be passed under the dorso lumbar junction as well as under the shoulders and hips in order that the spine shall remain in the neutral position. The victim who is lying face downwards with a simple wedge or comminuted fracture may be lifted quite safely in this position even without special slings for



FIG 48

Dangerous method of transport. The spine is forced into flexion and if there is a fracture or fracture dislocation the displacement must be increased.

the spine is then extended and the displacement reduced. The patient may be allowed to remain prone on a stretcher or in bed. If pillows are placed beneath the upper chest and head the position is perfectly comfortable.

Importance of initial radiograph—Before any attempt is made to reduce the fracture good radiographs must be examined in order to be sure that the case is suitable for hyperextension treatment. There are a few rare vertebral body injuries in which this treatment is inadvisable or even dangerous (Chapter XV).

Anæsthesia—An anæsthetic is not essential. There may be discomfort in the arms where the weight of the trunk is borne and in the abdominal muscles which are stretched but there is seldom severe pain in the back. Local anæsthesia has been advocated by injecting 20 c.c. of 5 per cent novocaine deeply between the spinous processes in the hope that it will diffuse through the fracture hæmatoma¹⁶. It is somewhat doubtful whether an impacted crush fracture of a vertebral body can really be anæsthetised in this way. In any event there is seldom pain in this region so that local anæsthesia is of little or no value. If the patient is a strong muscular individual who cannot relax spinal anæsthesia may be used. Morphine should be given sparingly for it may cause abdominal distension and predispose to paralytic ileus. General anæsthesia is a disadvantage and is only to be considered in fractures which are more than two or three weeks old.

Preliminary preparations—Reduction of the fracture and application of the plaster should occupy no more than ten or twelve minutes. All preparations must be completed before the patient is in position. A double layer of 8 in. wide stockinet is pulled over the trunk and fixed over the shoulders and in the perineum. There must be no bulky wadding. Even with a minimum of padding the plaster jacket will become loose within a few weeks and if wool and felt padding is used too generously the fracture will redisplace despite the plaster. Care must be taken to prevent pressure sores over the anterior superior iliac spines and especially over the kyphos. A pad of $\frac{1}{8}$ in. adhesive felt is placed over each iliac spine and a piece of $\frac{1}{8}$ in. adhesive felt 6 in. by 4 in. is applied over the spinous processes. A window 4 in. by 2 in. will be cut in the plaster centred on this piece of felt so that pressure over the kyphos is entirely relieved.

A pail of water, a supply of wide plaster bandages and a dozen dry plaster slabs are prepared to facilitate rapid application of the cast. Two tables are arranged end to end with a space between slightly greater than the length of the patient's trunk. The front table should be 10 or 12 in. higher than the other.

Position of the patient—The patient is lifted into position and he supports himself on the front table by his abducted arms (Fig. 489). It is important to note the exact position of the tables. The space between them must be so wide that the trunk is entirely unsupported. If the lower table extends beyond the symphysis pubis and the groins the pelvis cannot tilt forwards and the lumbar spine will not be completely extended. This is a frequent source of imperfect reduction (Fig. 489). The upper table must not be too

¹⁶Blair The Treatment of Fractures, Bristol 1934, 4th ed. 1st ed.
¹⁷Marique L'anesthésie pour la réduction des fractures dorsales. Jour de Chir. et Ann. de la Soc. Lép. 9 th Feb. 1937.

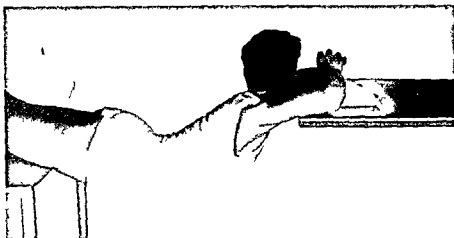


FIG 488

Correct position for postural reduction. The lower table extends to the upper thighs, the upper table is clear of the chest.

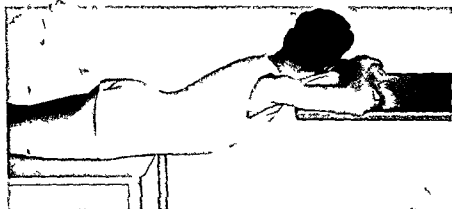


FIG 489

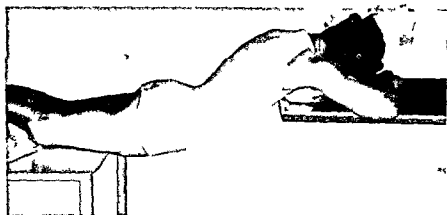


FIG 490

Incorrect positions for postural reduction. If the lower table extends beyond the groin, the pelvis does not tilt forwards and the lumbar spine is not fully extended (Fig 489). If the upper table is too near the chest, the plaster cannot be carried high enough and the dorsal spine is not fully extended (Fig 490).

close to the chest (Fig 490) The whole of the front of the trunk must be easily accessible so that the plaster can be carried up to the level of the clavicles

Application of plaster jacket—There is no necessity for any pause the fracture will be reduced by the time the plaster is completed Successive layers of plaster are rubbed firmly into each other This degree of pressure is sufficient to complete the reduction There should be no manipulation and no forcible thrusting over the kyphos The jacket must extend from the symphysis pubis and the groins up to the clavicles A strong rim of plaster must remain over the symphysis but an ellipse may be cut away from each groin to permit flexion of the hips Plaster may also be removed

from below the shoulders and axillæ in order to permit arm movement it must not be cut away from the clavicles or sternum (Figs 491 492) Only if there is an intact curve of plaster from the pelvis to the clavicle is flexion movement prevented Posteriorly the plaster extends from the gluteal cleft to the scapula

It is a common mistake to carry the plaster no higher than the nipple line Redisplacement is then inevitable If the patient complains that the plaster digs into the thighs or presses on the neck it must not be cut away so freely that it is possible for him to sit bolt upright and to flex forwards over the upper margin He must be taught how to sit and to be propped with pillows so



Fig 491

Correctly applied plaster extending from the groins and symphysis pubis to the clavicles The lumbar spine cannot be flexed



Fig 490

Incorrectly applied plaster It is cut too high at the groin and too low over the chest Displacement of the fracture will recur

that his trunk is extended away from the plaster Figs 493 494 show a plaster jacket which is no more than an ornament It is so short that it is not really immobilising the spine and it is certainly not preventing flexion movement The large abdominal window has been cut before the plaster was dry and the patient can flex the spine almost as freely as if he was not in plaster at all

Indications for cutting an abdominal window are (1) respiratory distress in emphysematous patients who rely on abdominal respiration (2) abdominal distension causing flatulence and vomiting A window should not be cut as a routine and unless there is some urgent indication it should never be cut before the plaster is thoroughly dry

For the first few hours the patient lies with pillows beneath the convexity of the arched spine He may then be turned on to the

The application of a body cast partially in hypertext or general all cases will prove a good result. A plaster cast may occur unless relief is afforded at once. It is often not built in time to permit of a position of the patient and therefore a very large plaster

face with pillows beneath the thighs and the upper chest. Thereafter the position is changed every few hours in order to avoid pulmonary congestion. The next day the patient may sit up and within a few days walking is resumed.

Radiographic control of reduction—Perfect recovery is possible only if perfect reduction is insisted upon. Even slight degrees of wedging of the vertebræ may cause persistent aching pain. Post reduction lateral radiographs are essential and there must be no hesitation in applying a new plaster if the reduction is not absolutely complete. The anterior vertical depths of all the vertebræ should be no less than the posterior depths. Moreover the intervertebral disc spaces should be fully opened. If this has not been accomplished the anterior common ligament is not taut and redisplacement will arise. The standard of reduction which is often accepted

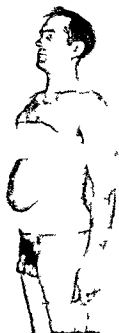


FIG 493



FIG 494

A worthless plaster jacket. This type of plaster accounts for many of the imperfect results of treatment of vertebral fractures. (By courtesy of *Jour Bone and Joint Surg.*)

is far too low and it is for this reason that persistent backache and incapacity from heavy work is still so common after simple spinal fractures. There is no justification for failure to secure perfect reposition of simple wedge fractures (Figs 493, 496).

After-treatment—Exercises throughout the period of immobilisation—In uncomplicated cases exercises for the spinal and abdominal muscles are practised regularly for not less than five minutes every hour of every day. While lying on the face with the arms by the side, the head and shoulders are slowly raised from the bed and lowered again (Figs 501, 502). Each hip is slowly hyperextended with the knee straight and as the patient becomes more expert both hips are simultaneously hyperextended. These exercises are far more effective in maintaining the tone of spinal muscles

than any direct faradic stimulation and massage even if such treatment was possible. The strength of the spinal musculature at the end of four months immobilisation in plaster should be very much greater than it was before the fracture, the tone should be quite perfect, the muscle bellies should stand out like bars of iron.

Not only does regular exercise maintain the tone of the spinal musculature and make it quite unnecessary to wear a spinal support in the later stages but it prevents stiffening of the immobilised joints of the spine and it restores the confidence of the patient in his functional recovery. Within a few days of injury walking is resumed. The patient should dress normally, go into



FIG. 495



FIG. 496

Wedge fracture of vertebral body (Fig. 495). Eleven months after postural reduction and plaster immobilisation (Fig. 496).

the streets into the parks and pursue normal activities. The sooner a man is dispossessed of the idea that his back is broken and that 'he will never walk again' the more certainly will functional disturbances be avoided.

Check X-ray and new plaster—After four weeks a further radiographic examination is made. If the films show that there is a tendency to recompression of the vertebral body, or if the jacket is obviously becoming loose, a new plaster must be applied in maximum hyperextension.

Duration of immobilisation—In many cases of fracture of the spine the plaster jacket is removed too soon. Crush fractures are slow in consolidating, and flexion movement must not be allowed earlier than four months after injury. Severely comminuted fractures unite even more slowly, and in these cases it may be necessary to retain the jacket for six months.



FIG 497



FIG 498

Comminuted fracture of vertebral body (Fig 497) Post reduction X ray shows complete replacement of marginal fragments and full opening of intervertebral disc spaces.



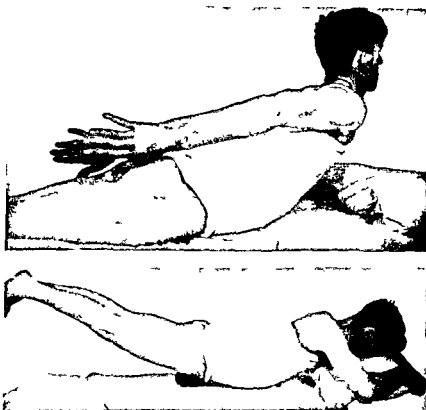
FIG 499



FIG 500

Fracture dislocation of vertebrae with comminuted fracture of lower vertebral body and paraplegia due to cauda equina injury (Fig 499) Post reduction X ray shows complete reduction of dislocation and almost complete replacement of marginal fragment (Fig 500) The paraplegia recovered fully (H Isaac Comminuted Fracture of Spine with Paraplegia *Br J Med* Jan 193 1 83)

Convalescent brace—It is inadvisable to use a posterior spinal support at any stage of treatment. In the early months it gives the surgeon a false sense of security. However skilfully made and however heroically worn, there is no type of posterior support which will prevent collapse of an unsoundly consolidated vertebra. In later months it gives the patient a false sense of security. Months or even years may elapse before he can be persuaded to discard a splint which has done nothing except prevent the stretching of adhesions¹. Either the fracture is not firmly consolidated



FIGS. 501-502

Exercises for the spinal and abdominal muscles are practised regularly throughout the period of immobilisation.

and a posterior splint is inadequate or the fracture is consolidated and a splint is unnecessary. But it is understating the case to say that a posterior spinal support is unnecessary; it is actually harmful. The fitting of a steel and leather splint suggests to the patient that the surgeon is not confident of the result and that he is still afraid. The fear which is in the patient's mind is magnified. He is more than ever convinced of the dangerous nature of his injury. The splint constantly reminds him of his back and psychological disturbances very often increase. It is very much better to continue the immobilisation in plaster until the surgeon is entirely satisfied, so that he can assure the patient that his back is perfectly safe and that pain and discomfort are due only to adhesion formation which will disappear if exercises are continued.

¹Tucker. Treatment of Fractures of Spines. *Virginia Med. Month* 1934, liv, 154.

Mobilisation exercises—As soon as radiographs show firm union the plaster is discarded. If exercises have been practised regularly and functional activity has been maintained it will be found that the immediate range of movement is surprisingly free. As soon as the plaster is removed the patient can reach with his outstretched hands to below the level of the knees and with suitable exercises a normal range of movement is regained within two or three weeks. It is quite untrue to suggest that immobilisation in hyperextension causes permanent stiffness. Even in middle aged and



FIG 503

Exercises are continued in the gymnasium. This patient who is climbing wall bars is approaching the end of his four months' immobilisation as shown by the looseness of the plaster jacket.

elderly patients the spinal joints are rapidly mobilised (Fig 507). Manipulation for adhesions is very seldom required.

Strengthening exercises—Treatment is not concluded when the fracture is united nor when full movement is restored but only when normal power has been regained with the ability to sustain vigorous exercise and strenuous work without discomfort or fatigue. Weakness has been minimised by active extension exercises practised regularly throughout the period of immobilisation but heavy manual workers such as miners who often sustain fractures of the spine need still more power. Exercise must be continued in the gymnasium with the aid of medicine balls, wall bars and pulleys and weights (Figs 503-506). Swimming, football, bowls and other recreations are encouraged. Within about two months of discarding the plaster recovery should be quite complete.

Treatment of old compression fractures—The ideal time for the reduction of a fracture of the spine is the first two or three days after injury. Perfect

reduction is still possible during the next two or even three weeks but after that time compression fractures are reduced with difficulty. If the fracture is from two to ten weeks old when it is first seen manipulative reduction should be attempted under anaesthesia. If general anaesthesia is used the Davis technique may be found more convenient than the Watson Jones

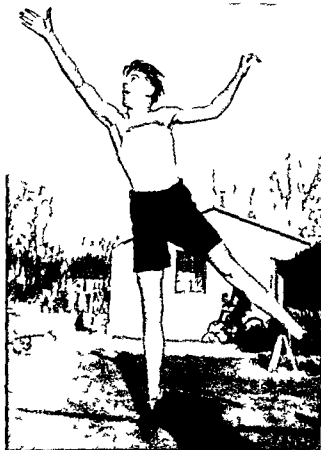


FIG 504

This patient never stops to think that his back is broken. He is playing deck tennis and is too full of the joys of life to develop functional complications.

him from regaining the muscle control on which his future depends.

Results of treatment of compression fractures.—When the reduction of spinal fractures by hyperextension was first introduced in 1930 there was some controversy as to the merits of the technique.¹ It was said that perfect reduction was not possible—that if it was possible it could not be sustained and that if it could be sustained the result was no better than if it had never

There was similar controversy in Germany where Bollinger defined it as the first time and in France where discussion continued until 1938 when Lillie and Loeferer presented non-surgical treatment of fractures due to rickets. Parkes, Miss, etc. A Lancet review of this work (Lancet i 1939) says: "This monograph presents a study of the results of a reduction of fractures by hyperextension and fixation in hyperextension according to the methods familiarly used by Watson Jones and Lillie and Loeferer. The controversy with this monograph is to settle whether or not the results of the hyperextension or manipulation reduction by manipulation in hyperextension—has already been settled in the literature in the American literature of the success of the treatment of certain fractures."

technique and the hyperextended position should be sustained for five or ten minutes before plaster is applied. It is seldom necessary to use direct pressure over the lophos. Even if wedging is imperfectly corrected the jacket should be retained until four months from the date of injury in order to prevent further wedging of bone to enable ligaments to tighten and to encourage redevelopment of the spinal musculature. If the injury is more than ten weeks old it is unlikely that hyperextension of the spine will reduce the compression of the vertebra and treatment should be concentrated on intensive redevelopment of the spinal muscles. The worst treatment of such a case is to apply a spinal brace or support which will not reduce the fracture will not prevent the bone from wedging and will do nothing except increase the patient's fear and prevent

been achieved I dealt with these criticisms in a review of end results published in 1938¹ which may be summarised as follows —

Anatomical results—It is always possible to correct wedging and compression in recent lumbar fractures. It is easy to secure a reduction so accurate that the level of injury cannot be localised even with the aid of radiographs. With careful after treatment reduction can be maintained. In no more than a small percentage of cases is there measurable compression of the bone in the final radiographs still less is there kyphotic deformity which is evident clinically.

Spur formation—The formation of bone spurs at the anterior margins of vertebral bodies is well marked only in unreduced fractures. Large spurs represent unreduced fragments of the fractured vertebra. They are commonly present in untreated cases but seldom in accurately reduced fractures. Slight spur formation may follow the best treatment. It is due to subperiosteal ossification at the attachment of the anterior common ligament. It does not indicate arthritic change. It is not a source of pain and it is of no clinical importance.

Narrowing of intervertebral disc—Narrowing of the discs after simple compression fracture is rare. It is more frequent in comminuted fractures because the disc is often ruptured. Slight narrowing of the disc is unimportant but it may account for a trace of kyphosis even when there is negligible deformity of the bone.

Functional results—There is no difficulty in regaining normal movement of the spine even after immobilisation for many months. In two thirds of cases there is complete freedom from pain and patients report that their backs are as good as before the accident, one third complain of occasional discomfort or pain. This freedom from symptoms compares very favourably with the results of uncorrected wedging where pain is almost monotonous in its regularity. Years after injury, long after the conclusion of medico-legal dispute many patients complain of aching pain which is presumably due to mal alignment of the interarticular joints.

Capacity for work—Of the patients under review including both simple compression fractures and comminuted fractures 80 per cent resumed their

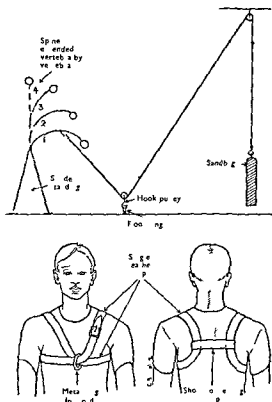


FIG. 50.

Resistance pulley and weight exercises for spinal injuries after removal of the plaster as developed by Nicol (B. J. Med. Jour. 1941, 501)

¹ Watson Jones. Lost spinal Reduction of Fractures of the Spine. Jour. Bone and Joint Surg. 1938, xx, 67

pre accident employment one half were men engaged in heavy manual labour The average incapacity period was ten months in heavy workers and seven months in patients engaged in lighter employment

Later review of results in 1943—Later investigation shows the importance of distinguishing simple compression fractures and comminuted fractures In the 1938 review where these injuries were grouped together it was found that two thirds of the patients were completely free from pain and about the same proportion resumed heavy manual labour But such a conclusion was

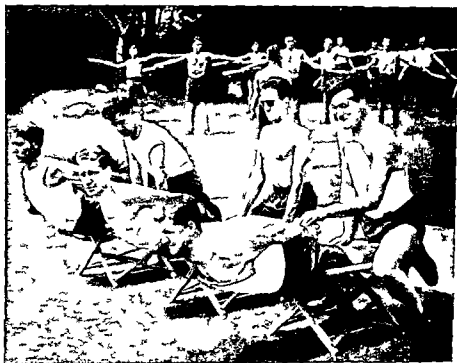


FIG 506

Six patients with fractures of the spine assisting each other to redevelop the spinal musculature Such group work encourages a spirit of competition and rivalry It is the lone patient left to himself who becomes introspective (A series of patients under treatment by Spinal on Leader II Canto)

misleading because the prognosis is very different in the two groups When simple wedge compression fractures are treated by reduction of the deformity, protection in plaster and early active exercise perfect recovery should be secured in many more than two thirds of the cases whereas many less than this make a complete recovery when comminuted fractures are similarly treated All the success claimed for the postural reduction of spinal fractures has been confirmed in the case of wedge compression fractures Recovery is complete movement is normal there is freedom from pain and if exercise and rehabilitation have been sufficiently active the man should come out of plaster at the end of four months with better muscles and better posture than he had on the day of injury On the other hand in the case of comminuted fractures the result of conservative treatment has been less satisfactory The treatment of these fractures should be revised

TREATMENT OF DORSO-LUMBAR FRACTURES—COMMUNUTED TYPE

A comminuted fracture of the dorso lumbar spine is a very different injury from a wedge compression fracture the mechanism of injury is different the gravity of injury is different the prognosis is different and it is now clear that treatment is different Whereas simple wedge fractures are due to vertical compression dispersed over several segments comminuted fractures are due to a sharply localised angulating force Not only is the bone grossly comminuted by impact of the anterior angles of

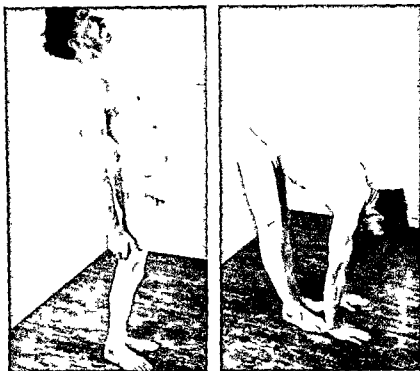


FIG 507

Range of movement in a man aged fifty five after a crush fracture of the spine immobilised in hyperextension for five months Even in the elderly patient, immobilisation in hyperextension does not cause permanent stiffness

vertebral bodies above and below but the intervening intervertebral discs are ruptured The very fact that the angle of an adjacent body has ploughed into the middle of the fractured body shows that at the moment of injury there was such a degree of hyperflexion deformity that interarticular joints must have been dislocated or pedicles fractured The force may be so great that fragments of the body are displaced backwards into the neural canal It is obvious that an injury which causes local disruption comminution of bone rupture of discs fractures of pedicles tearing of ligaments dislocation of interarticular joints encroachment on foramina pressure on nerve roots, and sometimes involvement of the spinal canal is likely to cause persistent aching and discomfort even when displacement is reduced There is no doubt that displacement can be reduced by the same postural technique which is successful in wedge compression fractures The anterior common ligament is usually intact and when it is put on stretch by hyperextension

of the spine displaced fragments are replaced with surprising accuracy. Moreover the position can be maintained by a closely fitting plaster jacket and so long as the plaster is worn there is no recurrence of pain. But the fracture is very slow in consolidating and immobilisation must be continued for at least six months. Even then it may be found that when the plaster is removed root pain recurs and the cast must be re-applied. Sometimes it has been necessary to re-apply the jacket several times and to continue protection for as long as nine to twelve months. The problem may still not be solved because rupture of intervertebral discs with arthritic change in intervertebral and interarticular joints often causes persistent aching pain which is not relieved until bony ankylosis finally takes place between adjacent vertebral bodies. Unfortunately the development of spontaneous ankylosis is slow and persistent pain, discomfort and weakness which persist over a period of several years may produce so deep a psychological impression that patients can never satisfy themselves that recovery is complete.

Indication for spinal fusion.—The conclusion must be accepted that whereas functional recovery after a simple wedge fracture is achieved within a few months by restoration of normal anatomical relations, functional recovery after a severely comminuted fracture may be achieved only after one or two years by ankylosis of vertebral bodies. If comminution is severe and it is evident that bony ankylosis is the optimum end result, the sooner the ankylosis is secured the better. The ideal treatment is to ankylose the laminae and spinous processes by a spinal fusion operation performed as soon as the patient's general condition will permit.

Pre-operative localisation of fractured vertebra.—No surgeon should embark on a spinal fusion operation without first localising the spinous process of the injured vertebra. Only three vertebrae must be fused—the fractured vertebra, the one above and the one below. Not only is it unnecessary and inadvisable to stiffen the spine by fusing more than three vertebrae but extensive fusions often fail and long grafts are often fractured or absorbed exactly at the level where ankylosis is most important. It is almost impossible to count the spinous processes accurately from the level of the iliac crest and it is certainly impossible to count them from the position of the twelfth rib or the angle of the scapula. I have seen many examples of spinal fusion even for tuberculous disease where the affected vertebra has been missed altogether. If this is possible in spinal disease where there is usually a well marked kyphosis, how much more is it likely to occur in spinal fracture where there should be no deformity. Two devices are available: (1) a few days before operation a small screw is driven into a spinous process near the level of injury through a tenotomy stab wound; lateral radiographs are taken, the position of the screw is identified at operation and from this known level the spinous process of the injured vertebra is localised. (2) immediately before operation a sterile mixture of lipiodol and lampblack is injected alongside one spinous process near the level of injury. The lipiodol shows the level in radiographs and the lampblack is easily seen at operation. The spinous process of the injured vertebra is counted up from this known position.

Technique of operation.—Through a midline incision the spinous processes and laminae are exposed by subperiosteal stripping. Haemorrhage is controlled by (1) keeping strictly to the subperiosteal plane and never dissecting into muscle masses, (2) picking the whole wound with small

gauze strips only one of which is removed at a time, as soon as dissection is completed in that area the pack is replaced before the next is removed. With these precautions the operation is almost bloodless, whereas without them hæmorrhage may be so profuse that the surgeon works blindly and may cause serious damage. When the three vertebræ are exposed, the spinous processes and laminae are cleaned with a sharp periosteum elevator. Fragments of bone are then turned up with a narrow gouge from the side of the spinous processes and the cortex of the laminae left attached at their bases and interlocked across the back of the ligamenta flava, thus forming a continuous mass of living bone chips. It is not essential to expose the interarticular joints and remove the cartilage as in the Hibbs' technique, but the fusion should be supplemented by one or preferably two, grafts cut from the crest of the ilium (or from the tibia) and laid alongside the raw spinous processes. The grafts need not be fixed by sutures or screws, when the muscle masses are stitched back into position they lock firmly into the bed of raw bone and sound fusion can be relied upon.

Post-operative treatment—A plaster jacket may be applied immediately, but as a rule it is safe to defer the application of plaster for a few days. The spine should be in the extended but not fully hyperextended position. Plaster immobilisation is continued for a period of four to six months, recumbency being advisable for the first two months. Spinal muscle exercises should not be practised with usual vigour until the operative fusion is almost sound. During the last few weeks of immobilisation in plaster the intensity of exercise is rapidly increased and after the fifth or sixth month full rehabilitation is instituted with energetic exercise games and gymnastics inspiring treatment and every measure which will restore the patient's confidence. No posterior spinal support should be worn, it is useless and it is harmful—it interferes with the necessary exercise, disturbs the patient's confidence and undermines his morale. *The best spinal brace in the world is the musculature of the patient's own spine, the muscles should be so powerfully developed that it would be an obvious insult to add the support of a spinal brace.*

TREATMENT OF HIGH DORSAL FRACTURES

The dorsal spine forms the arm of a lever which has its fulcrum at the dorso lumbar junction. The strain of forcible flexion movement is therefore transmitted to the level of the last dorsal and first two or three lumbar vertebræ, and fractures nearly always occur at this level. But although in civilian life high dorsal fracture is rare, in time of war it is often sustained by pilots of fighter aircraft. The incidence of high dorsal fracture in the Royal Air Force is almost as great as the incidence of lumbar fracture. The reason for this unusual localisation lies in the special harness worn by pilots to permit aerobatics, and in the event of crash landing to prevent an uncontrolled forward fling of the trunk with violent impact of the head against the instrument panel of the aircraft. If the pilot is wearing a simple waist belt and makes a crash landing the trunk swings forwards with momentum, and there is violent flexion at the usual level of the dorso lumbar junction, but if he is wearing the special harness which passes in front of both shoulders and holds the trunk back to the seat, only the pilot's head flings forwards and the site of angulation is raised from the lumbo dorsal junction to the

upper dorsal level. As a rule the injury is a simple wedge compression fracture with no comminution or dislocation.

Fractures at this level are very difficult and often impossible to reduce. The success of postural reduction of fractures at the dorso lumbar junction lies in the tremendous leverage of the segments of spine above and below the fracture which lies exactly at the fulcrum. But a high dorsal fracture lies in the middle of one of the arms of leverage and not at the junction of the two. No method of hyperextension has been found completely successful in reducing the compression. But the very factor which makes reduction difficult makes it unimportant. Since the injury is in a relatively immobile segment of the spine uncorrected wedging or compression seldom causes persistent pain.

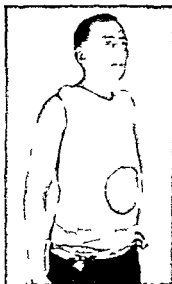


FIG 508

Plaster jacket for high dorsal fracture. Even more than usual reliance is placed on muscle exercise.

A plaster jacket is applied with the spine in hyperextension, either by the two table or the foot suspension method or perhaps more conveniently by the motor jack technique (Fig 486). A wing of plaster is then added on each side, following the line of the clavicles to the front of the shoulder joints which are fully braced back (Fig 508). Despite the high level of fracture it is then unnecessary to include the head and neck in the plaster. Even more than usual emphasis is placed on the importance of muscle exercise, the erector spinae must be so developed and become so powerful, that it fulfils the function of a surgical brace. The plaster is discarded in about two months and functional

recovery is complete within three or four months.

WAR FRACTURES OF THE SPINE

A surgeon will be called upon to treat at least twenty simple fractures of the spine for every fracture with paraplegia. This is true not only of the injuries of peace but also of the casualties of war.¹ Penetrating wounds due to bullets, shell fragments and bomb casing are rare, fractures due to compression and hyperflexion are common. There is often little proportion between the degree of hazard and the severity of fracture. For example, two airmen sustained compression fractures of the spine on the same day, one had fallen off a building in the black-out and crashed 60 ft., the other had been crossing the landing ground and sat down heavily in the mud, the second was more severely injured than the first. Nevertheless in each military service there are special risks with which fracture of the spine is associated.

Spinal fractures in air crews—Fractures due to crash landings are discussed on p. 321. If the airman is a fighter pilot wearing protective harness, there is likely to be a compression fracture of one or more vertebra in the upper dorsal spine. If he is wearing a waist belt, or is a member of a bomber crew wearing neither belt nor harness, he will probably sustain a fracture or fracture-dislocation in the lumbar region. Air crew casualties are characterised by the multiplicity of their injuries. The hazards of flak

¹ R. Watson-Jones. War Injuries of the Spine. *Proc. Roy. Soc. Med.* 1941 xxxiv 454

over the target may be followed by machine gun bullet or cannon shell wounds on the return flight and finally by multiple injuries sustained in the crash landing of a damaged aircraft. Very often therefore fracture of the spine is accompanied by head injury, disruption of the pelvis, penetrating joint wounds, compound fractures and burns all in the same patient. The spinal fracture may be the least important injury and reduction must sometimes be deferred for several days or even for one or two weeks. Meanwhile the patient lies on a bed with fracture boards and with pillows under the lumbar spine.

Spinal fractures in paratroops—Although the rate of a parachute descent and the force of impact on landing depends on air currents, it is at least the equivalent of a 10 ft jump and if a man lands heavily on his feet he may sustain fractures of one or both os calcis and the spine. But it is unusual to make a strictly vertical descent. As a rule there is oscillation and the man strikes the ground with his buttocks either on a forward or on a backward swing. In either event vertical compression or flexion of the spine may cause a lumbar wedge fracture. The deformity is often minimal and paratroops are tough so that the diagnosis is sometimes missed for several weeks. Occasionally during a forward roll after landing the neck is acutely flexed and a fracture or fracture dislocation of the cervical or upper dorsal spine is sustained. When a pilot bales out during combat the risk of injury may be greater. One airman for example in baling out at 20 000 ft struck the tail of his aircraft and was rendered unconscious. He regained consciousness before landing to find that both shoulder joints were dislocated and the parachute harness was twisted round one knee from which he was suspended. The medial ligament of the knee joint had been ruptured finally because his landing was heavy he sustained a comminuted fracture of one os calcis and a compression fracture of the spine. Despite all these injuries he was flying once more within seven months.

Spinal fracture in tank warfare—Wedge fractures of the lumbo dorsal spine have occurred in tank crews due to the compression force of heavy jolting of the tank over uneven ground being transmitted through the flexed spine of a soldier sitting crouched in a confined space.

Spinal fracture in naval warfare—When a torpedoed ship is sinking a sailor may jump from the uppermost side of the deck but if he is to clear the bilge keel which now appears above the sea level the jump must be much wider than would appear necessary and in many cases men have landed not in the sea but on the bilge keel or ship's side thus sustaining fractures of the os calcis and spine. More frequently the sailor sustains this dual injury without even leaving the deck. The fractures are due to the explosion of a mine below the vessel. Instead of the victim crashing from a height and striking the deck the deck comes up and strikes the victim. In other cases the sailor is lying on his back and the explosion beneath the ship flattens his lumbar lordosis with such violence that one or more vertebrae in the middle of the curve are crushed.

Spinal fracture in air raid casualties—Fracture of the spine is a relatively unusual injury in civilian air raid casualties. A few patients are buried in debris and sustain forcible flexion of the spine with a comminuted fracture of the lumbar region. A certain number of dislocations and fractures of the cervical spine have occurred in demolished air raid shelters where the concrete roof of the shelter has fallen on the heads and shoulders of those within.

CHAPTER XX

VERTEBRAL FRACTURES WHICH MUST NOT BE HYPEREXTENDED

The usual types of lumbar and dorsal fracture which we have discussed in the last chapter are produced by flexion and reduced by hyperextension on they may be described as the safe fractures. They include about 90 per cent of vertebral body injuries. It is almost impossible to do harm and to achieve perfect reduction it is necessary only to reach the limit of hyperextension.

There is another group of relatively rare injuries which are the dangerous fractures. It is important to recognise them for hyperextension treatment may aggravate or even produce nerve compression and paraplegia. Four injuries are to be classified in this group.

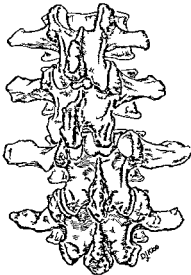


FIG 500

Locking of articular processes in lumbar fracture dislocation with lateral or rotatory displacement of the upper vertebra

- 1 Fracture dislocation with locking of the articular processes
- 2 Comminuted fracture with neural canal involvement (fracture of posterior wall of vertebral body)
- 3 Hyperextension fracture of the vertebral body or neural arch
- 4 Traumatic spondylolisthesis

Fracture dislocation with locking of the articular processes is the most common of this group of injuries. The other three types are of considerable rarity.

FRACTURE-DISLOCATION WITH LOCKING OF ARTICULAR PROCESSES

Dislocation of an intervertebral joint must be accompanied by a neural arch injury which is either a fracture of the articular processes or a dislocation of the interarticular joints. If the articular processes are fractured there is probably no obstacle to reduction by simple hyperextension. The same technique which

has been described for wedge and comminuted fractures may be cautiously employed and will usually be successful in reducing the dislocation.

If the interarticular joints are dislocated the situation is more dangerous. No attempt must be made to extend or hyperextend the spine until the relation of the articular processes has been determined. If the dislocation

of the upper segment is directly forwards and there is no lateral displacement or rotation the descending articular processes of the upper vertebra lie exactly in front of the space between the ascending articular processes of the lower vertebra. The displaced processes will probably slide back into position as the spine is extended. On the other hand if there is a lateral displacement of the upper segment or if the upper segment is rotated the positions of the articular processes no longer coincide. The descending process of the upper vertebra is actually locked in front of the ascending process of the lower vertebra on that side (Fig 510). If the spine is hyperextended the upper articular process cannot slide back even if traction is applied at the same time. The spine *hinges* backwards pivoting on the articular process. The vertebral bodies separate from each other the intervertebral space is abnormally widened and the spinal cord or cauda equina is stretched and



FIG 510



FIG 511



FIG 512

Lumbar fracture dislocation with locking of articular processes (Fig 510). If the spine is simply hyperextended the upper process cannot slide back the vertebrae are forced apart and the cord injury may be increased by stretching (Fig 511). It may be necessary to excise the lower articular process (Fig 512). (By courtesy of J. W. Bone and J. W. S. S. S.)

attenuated (Figs 510 511). Paraplegia may be aggravated or even produced by the treatment.

Reduction by open operation—These dislocations must be reduced by open operation and no attempt must be made to hyperextend the spine until the locked articular processes are exposed. Two operative procedures are available. The ascending articular process of the lower vertebra on the side towards which the spinous process of the upper vertebra is displaced or rotated may be excised at its base.¹ The obstacle to backward sliding of the upper segment is then removed and as the spine is gradually hyperextended the dislocation is reduced safely. Rogers² has described successful operative reduction without excision of the articular process. The case was one of dislocation at the first lumbar level with incomplete paraplegia. Attempted manipulative reduction by traction and hyperextension failed to unlock the articular processes and caused immediate increase in the paralysis which

¹ Minto and Irwin *Brit Jour Surg* 1938 xxv 621

² Watson Jones *Jour Bone and Joint Surg* 1974 xx 533

³ Rogers *Jour Bone and Joint Surg* 1935 xx 691



FIG 513

Radiograph of lumbar fracture dislocation with locking of articular processes. Before the spine is hyperextended the processes must be disengaged by open operation.



FIG 514

Fracture dislocation of lumbar spine with lateral angulation and interlocked articular processes on the right. After facetectomy on this side the dislocation was reduced. (Operated on by W/103 Commander J. L. Duck.)

became total. The same day under local anæsthesia the articular processes were exposed. Cautious flexion of the spine by lowering each end of the operating table freed the processes which were then rotated into alignment with a periosteum elevator. Hyperextension completed the reduction. There was full recovery from the paraplegia and a perfect functional result.

HYPEREXTENSION FRACTURES OF THE LUMBO DORSAL SPINE

Fractures of the spine produced by hyperextension are extremely rare. If they are treated by the usual method which is applicable to flexion fractures the displacement will be increased.



Fig 15

Comminuted fracture due to hyperextension. The anterior marginal fragments are avulsed by the anterior common ligament.



Fig 16

Hyperextension fracture of both pedicles of the first lumbar vertebra sustained spontaneously by an acrobat dancer while on the stage.

Comminuted fracture—The attachment of the fibro cartilage and of the anterior ligament remains undamaged but the strain transmitted through these structures breaks up the bone. The anterior half of the body separates into two quadrants, an upper which is tilted and displaced upwards and a lower which is tilted downwards. The posterior half of the body remains intact. In the case shown in Fig 15 a plaster jacket was applied with the patient sitting on a stool and recovery was uneventful.

Fracture of pedicles—An acrobatic dancer took up her profession at such a late age that it was always necessary for her to spend from ten to fifteen minutes loosening and limbering before going on to the stage. One night she was called on hurriedly. In the course of the dance she slowly hyperextended the spine until her head was between her knees. There was an ominous crack and an agonising pain. She had fractured both pedicles of the first lumbar vertebra (Fig 16). Despite many months of immobilisation



FIG 51

Applicat on of plaster jacket w th head tract on. The plaster may also be applied with the patient sitting up in his bed which is wheeled under the head suspens on apparatus.

the fractures failed to unite by bone. A firm fibrous union developed. She now pursues all normal recreations without pain or discomfort but she has not resumed professional dancing.

Fractures of the pedicles and articular processes must be differentiated from the congenital fissures which may be seen in the neural arches of the vertebrae especially in the lumbar region.¹³

COMMUNUTED FRACTURE WITH NEURAL CANAL INVOLVEMENT

Communited fractures are due to acute hyperflexion with such sharp angulation that one vertebral body is impacted into the middle of the vertebral body below. As the intact vertebra ploughs into the fractured body it displaces anterior marginal fragments forwards. In very rare cases when the flexion is still more forcible it may drive posterior fragments backwards (Fig 518). The posterior wall of the centrum is fractured and the upper posterior quadrant is displaced backwards into the neural canal. The lumen of the canal is seriously narrowed and the margin of safety is reduced. Hyperextension of the spine may increase the cord pressure and cause paraplegia. At the same time it must be recognised that flexion of the spine which reproduces the original displacement is still more capable of producing paraplegia by forcing the displaced fragment still further back. Rogers⁴ has described a case of such a fracture which had been overlooked and left untreated in which paraplegia supervened two years after injury from the pressure of increasing deformity with excessive callus formation within the spinal canal.

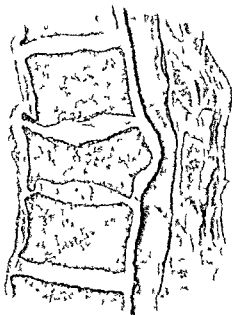


FIG 518

Comminuted fracture with encroachment on the neural canal due to backward displacement of posterior marginal fragment

A plaster jacket must be applied at once, but not by the usual hyperextension technique. The patient is sat upright, head traction is applied, and the spine is fixed in the extended but not in the hyperextended position (Fig 517). The jacket must be worn for not less than four months.

SPONDYLOLISTHESIS

Clinical features—Forward slipping of the fifth lumbar vertebra at the lumbosacral joint is usually associated with the congenital abnormality known as spondylolisthesis, in which there is a failure of bone fusion between the

¹ Steiner. Isolated Fractures of the Vertebral Arch. *Amer Jour Roent* 1938 xxxix 43.
² Rendich Westing. Accessory Articular Process of Lumbar Vertebra and its Differentiation from Fracture. *Amer Jour Roent* 1933 xlii 56.
³ Studdert Nichols. Ununited Anomalous Epiphyses of Inferior Articular Processes of Lumbar Vertebra. *Jour Bone and Joint Surg* 1933 xv 591.
⁴ Rogers. *Jour Bone and Joint Surg* 1938, xx 690.

vertebral body and the neural arch^{1*} The developmental defect is seen less commonly in the fourth lumbar vertebra and the fourth vertebral body slips forwards on the fifth When there is no bony continuity between the body and the posterior arch, stability depends entirely on the ligamentous structures There is impairment of the normal resistance to static and dynamic stresses, and injuries or even strains of a minor character may then cause forward dislocation Friberg who has had the unusual opportunity of investigating more than one hundred cases of complete spondylolisthesis, has shown that the displacement usually occurs at the age of eleven or twelve

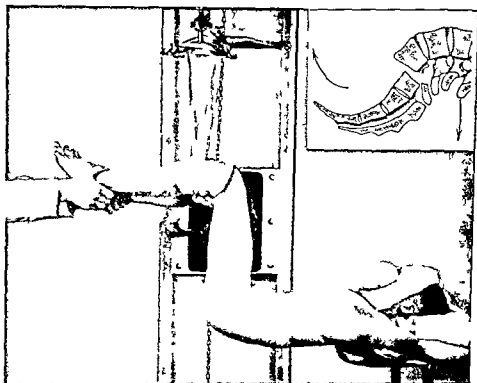


FIG. 19

Method of reduction of spondylolisthesis appl cable in adolescents where slipping is recent

Slipping may continue for a few years before the dislocated position is finally stabilised In the most severe cases there is gross deformity Lordosis becomes so extreme that the lumbar region sinks into the pelvis and the trunk is disproportionately shortened When viewed from the side, the thorax and spine lie entirely in front of the plane of the lower limbs The capacity of the pelvis is often greatly reduced and this may be of serious significance in child bearing women There is persistent aching pain in the back an unwillingness to stoop and an inability to lift weights In some cases paralysis develops from stretching or compression of the roots of the cauda equina

Treatment—Many operations have been performed to stabilise the vertebra in its dislocated position by posterior bone grafts^{3,5} Reduction

* Brailsford Lumbo-sacral Deformities *Brit Jour Surg* 1909 xvi 502 *Brit Jour Pediatr* 1909 ii 344
 * Pfahler and Vastine Spondylolisthesis and "spondyloschisis" *Surg Gyn Obst* 1934 lxxvii 691
 * Ryerson Spondylolisthesis with Paralysis Bone Transplantation *Jour Amer Med Assoc* 1915 lxi 24
 * Albee Spondylolisthesis *Jour Bone and Joint Surg* 19 15 40
 * Hibbs Lumbo-sacral Abnormalities *Surg Gyn Obst* 19 2 xlviii 604

of the displacement has seldom been attempted. With one exception¹ the attempts which have been made have failed. There are two reasons for this failure. Reduction is possible only within a few months, or at the latest within a year or two of the time of actual dislocation of the vertebra. Successful reduction is probably impossible in patients over the age of fifteen. Furthermore reduction cannot be achieved by hyperextension of the spine. Hyperextension increases the lordosis and it increases the inclination of the sacrum until it is more nearly horizontal and the axis of the lumbo sacral joint vertical. In this position the forward slide of the fifth vertebra must increase. The posture which is adopted subconsciously by the patient is the exact opposite of this. In his attempt to relieve the back strain he



FIG 520



FIG 521

Spondylolisthesis in a girl aged twelve (Fig 520). After manipulative reduction and operative fusion of the spine the position is considerably improved. The extreme lordosis is corrected, pelvic obstruction is relieved and pain in the back and leg is cured (Fig 521).

tries to reduce the lumbar lordosis. He stands with the knees and hips flexed so that the pelvis is tilted back, the sacrum is therefore held more vertically and the lumbo sacral joint is more horizontal. This subconscious posture gives the key to manipulative reduction. The lumbar lordosis must be flattened and the hips must be flexed.

Manipulative reduction—One case has been reported in which attempted manipulative reduction improved the position. Jenkins¹ treated a severe spondylolisthesis in a boy of fifteen by the gradual traction of a sling under the pelvis passing to an overhead beam. The hips were slightly flexed and traction was applied to the lower limbs from the foot of the bed.

The method which I have employed has not been previously reported. It is illustrated in Fig 519. The patient is anaesthetised. The head and shoulders are supported on the edge of a table. The hips are flexed to the right angle and vertical traction is then applied so that the lower limbs and pelvis are pulled upwards. The position obliterates the lumbar lordosis, the vertical traction pulls the sacrum forwards and body weight pulls the

¹ Jenkins. Spondylolisthesis. *Brit Jour Surg* 1936 xlii 80.

lumbar vertebræ backwards. If necessary a weight may be suspended across the abdomen in order to increase this backward pull. A double plaster spica is then applied with the hips still flexed to the right angle. Fig 520 shows how almost completely a severe degree of spondylolisthesis may be corrected in a girl aged twelve.

Operative fusion—Redisplacement must be prevented by operative fusion of the lumbo sacral region. A few weeks after the manipulative reduction a window is cut in the back of the plaster to expose the sacral and lower lumbar regions. A very thorough fusion of the articular processes, laminae and spinous processes of the last three lumbar and the sacral vertebrae is necessary. A graft from the tibia may be implanted, but a graft alone

must not be relied upon. The complete Hibbs operation, including excision of the interarticular joints, must first be performed.

Anterior bone block—Fusion of the fifth lumbar body to the sacrum by an anterior bone block introduced by the abdominal route was suggested by Capener¹ and first performed by Burns². It was devised because posterior fusion was believed to be mechanically unsound. In fact, however, if the dislocation is first reduced and if the bone graft is supplemented by a thorough bone fusion, the posterior operation is entirely satisfactory and the anterior graft has no structural advantage over it (Figs 522-523). The advantage of the anterior operation is

FIG 522 An anterior bone block (Fig 522) has little or no structural advantage over the less formidable operation of posterior fusion (Fig 523)

that fusion is confined to the lumbo sacral joint, so that mobility of the third and fourth lumbar vertebrae is unimpaired, and shortening of the lumbar region is not aggravated by arrested growth of three vertebrae. On the other hand, anterior fusion is a formidable operation. Of the eleven reported cases two died within a few days of operation.^{3,5}

In older patients where the bones have become stabilised in the displaced position no attempt should be made to reduce the deformity. The symptoms may be relieved by a spinal support or by operative lumbo sacral fusion.

Traumatic spondylolisthesis—In rare cases an identical displacement is produced acutely from a severe injury in a spine which was previously normal. The pedicles are fractured and the fifth lumbar body is dislocated forwards. Injury of the sacral nerves may cause saddle anaesthesia of the perineum and gluteal regions and paralysis of the rectal and bladder sphincters. Reduction may be achieved by the same technique as that described for congenital spondylolisthesis but the possibility of interlocking of articular processes and the necessity for facetectomy must be borne in mind (p. 127).

¹ Capener Spondylolisthesis. *Brit Jour Surg* 1931 xix 34
² Burns Operation for Spondylolisthesis. *Lancet* 1933 i 1235
³ Mercer Orthopedic Surgery. London 2nd ed. 59
⁴ Mercer Spondylolisthesis—new operation. *Edin Med Jour* 1936 xliii 545
⁵ Friberg Studies in Spondylolisthesis. *Acta Chir Scand*, 1939 lxxvii suppl. 17

CHAPTER XVI

FRACTURES AND DISLOCATIONS OF THE CERVICAL SPINE

"Broken backs and broken necks are neither so disabling nor so deadly as they were formerly thought to be"—S T IRWIN

The average patient fears a broken neck even more than he fears a broken back and the psychological disorders which may be aroused by this fear must be anticipated. The fracture is no more remarkable than any other injury. Whether there has been a nerve complication or not, the surgeon's manner should be the same as if the injury was a fractured wrist, he must be reassuring, optimistic and matter of fact. The patient must not learn of the narrowness of his escape. It is the very worst treatment to suggest to the victim that he was 'within half an inch of his life'. In the early days caution may be necessary, but after two or three months of immobilisation when the surgeon's anxiety is over, this caution must be abandoned. The continued wearing of a collar is valueless and it serves only to remind the patient of his fears.

Four groups of cervical injury may be differentiated: (1) crush fractures of the vertebral bodies, (2) sprains and subluxations of the interarticular joints, (3) dislocations of the interarticular and intervertebral joints, and (4) fractures and dislocations of the atlas^{1 4}

CRUSH FRACTURE OF CERVICAL VERTEBRÆ

Simple compression and comminuted fractures of the cervical vertebral bodies are less common than dislocations and fracture dislocations. The fifth, sixth and seventh vertebræ may be crushed by forcible hyperflexion injuries. If there is no interarticular joint dislocation, the fracture is treated as lumbar crush fractures are treated, the displacement is reduced by hyperextension and the spine is immobilised in plaster.

Treatment—A piece of strong wood 3 in wide is nailed to a wooden table so that it projects 10 or 12 in from the end. It is padded with wool (Fig. 524). The patient lies face upwards with the free end of the wood at the level of the cervico dorsal junction. The head is supported by an assistant in neutral rotation, and lowered until the neck is fully extended. There is no necessity to raise the chin and to tilt the head back. This movement occurs between the skull and the atlas and it does not improve the position of the fractured vertebra. The patient should be looking at the ceiling rather than at the wall behind him. The arms are held aside while plaster is applied from the pelvis to the top of the head. The plaster should extend over the forehead to the level of the eyebrows, and it may

¹ Brookes. Dislocation of Cervical Spine (report of 50 cases) *Jour Missouri Med Assoc* 1930, xiv, 579
² Langworthy. Dislocations of Cervical Vertebrae (report of 30 cases) *Jour Amer Med Assoc* 1930, xiv, 88
³ Brookes and Frierdrit. Reduction of Cervical Dislocations. *Arch Phys Therap* 1932, xiii, 463
⁴ Roberts. Fractures and Dislocations Cervical Spine (37 cases) *Jour Bone and Joint Surg* 1937, xix, 199

then be cut away beneath the chin so that movements of the jaw and larynx are facilitated. A window is cut over each ear. Patients become surprisingly tolerant of the discomforts and within a few days they may be up and about normally dressed. Immobilisation is continued for three months. After removal of the plaster normal function is rapidly restored by active exercises. A protective collar or splint is of no real value and moreover it may prove difficult to persuade the patient to discard it.



FIG 594

Reduction of cervical crush fracture. Plaster is applied over the head, neck and chest to the pelvis with the cervical spine fully extended. (By courtesy of *Journal of Bone and Joint Surgery*)

SPRAIN OF THE CERVICAL INTERARTICULAR JOINTS

A sudden twisting or jerking movement of the neck may strain the interarticular ligaments without actually displacing or dislocating the joints. There is local pain, movements are guarded by muscle spasm and oedema and hæmorrhage round the adjacent nerve roots may cause pain referred to the arm, forearm and hand.

Diagnosis—Strain of the interarticular joints must be carefully differentiated from subluxation or dislocation. If there is no displacement the lateral radiograph shows that the joint surfaces of each pair of articular processes are strictly parallel. *Lateral views must also be taken with the spine fully flexed.* An incomplete dislocation may be reduced spontaneously by extension of the neck and radiographs in this position are often misleading (see p 158 figs 282-283).

Treatment—If there is no subluxation plaster immobilisation is unnecessary. A simple felt surgical collar is worn for a few weeks. An adequate support may be improvised from a man's stiff double collar opened to its full width bound with wool and banded round the neck. Pain and stiffness sometimes persist owing to secondary adhesion formation especially when there was osteoarthritic change in the joints. In many cases of cervical osteoarthritis the pain referred to the arm and along the

occipital nerves to the head is due to adhesions rather than to active arthritis and it may be relieved by manipulation. It is for this reason that osteopaths cure headache by manipulating the neck. The osteopath does not differentiate occipital neuralgia from the headache of intracranial disturbances.

SUBLUXATION OF CERVICAL INTERARTICULAR JOINTS

Interarticular joint subluxation is more frequent and more serious than is generally recognised. It is a type of dislocation of the neck and the only feature distinguishing it from complete dislocation is that the articular processes have not actually overridden (Fig. 523). In many cases the displacement is spontaneously reduced by simple extension movement and for this reason the injury is often overlooked. There may be hæmorrhage round the cord, paraplegia and death apparently with no bone injury. The subluxation can only be excluded by taking radiographs in the flexed position. Even if there is no danger of cord injury failure to immobilise the joints for an adequate period may cause recurrent subluxation. Nerve root irritation and compression then lead to persistent disabling neuritis in one or both upper limbs.



FIG. 523

Cervical interarticular joint subluxation. In this case the injury is almost a complete dislocation, but the articular processes are not overriding and reduction is therefore possible by simple extension as in Fig. 524.

Etiology—The displacement may follow a severe injury such as a fall on the head or a dive into shallow water. In other instances the accident appears unimportant. I have seen two cases arise from an involuntary forward jerk of the head sustained by a passenger in a car which stopped suddenly and unexpectedly.

Radiographic diagnosis—Forward tilting of the upper articular process so that its joint surface is no longer parallel with the process below is sometimes obvious in routine lateral radiographs. The upper vertebral body may show slight forward displacement or there is narrowing of the front of the intervertebral disc. The displacement may be recognisable only when the neck is X-rayed in moderate flexion.

Treatment—Complete plaster immobilisation is essential. The neck is fully extended and plaster is applied by the technique shown in Fig. 524. Immobilisation must be continued for not less than two months. Referred root pain usually subsides within a few days. If pain recurs after removal of the plaster a collar limiting forward flexion may be worn for a few weeks and then discarded for increasing periods each day. Pain persisting after several months may be due to adhesion formation, but recurrent subluxation must be excluded before the neck is manipulated.

Recurrent subluxation of interarticular joints—If the injury is overlooked, or if it is treated without complete immobilisation in the early stages, the ligaments do not fully tighten. Recurrent subluxation arises with persistent local and referred pain. The displacement is shown if lateral radiographs are taken with the spine in flexion (Fig 526)



FIG 526



FIG 527

Recurrent interarticular joint subluxation. Three years after injury there is still severe root pain (Fig 526). After extension of the neck and operative fusion the symptoms were relieved (Fig 527).



FIG 528



FIG 529

Recurrent interarticular joint subluxation after operative fusion with tibial bone graft showing the range of movement which is possible (same case as Figs 526 and 527).

Treatment—Conservative treatment may be worthy of trial. The spine is immobilised in plaster in full extension for four months. If displacement and root pain recur after removal of the plaster, operative fusion and bone grafting is necessary. A graft from the tibia, the crest of the ilium or a rib, is implanted on to the laminae and bases of the spinous processes. The fusion

must be as localised as possible in order to minimise subsequent stiffness of the neck (Figs 528-529). After operation the neck and head are immobilised in plaster for a period of from four to six months, until there is firm bony consolidation.

DISLOCATION OF CERVICAL INTERARTICULAR AND INTERVERTEBRAL JOINTS

When forward displacement of the upper segment of the spine is more marked the articular processes on one or both sides slip forwards over the articular processes below. The processes are locked in this position and the dislocation cannot possibly be reduced by simple extension movement. In many cases the lower vertebral body is crushed or an anterior marginal fragment is broken off and displaced forwards. As a rule the spinal cord is contused or compressed and there is paraplegia.

Treatment—It is useless to attempt the reduction of a complete unilateral or bilateral interarticular joint dislocation by simple extension of the spine. The various manipulative procedures which have been described^{1,2} also fail in many cases. Strong traction is necessary. It must be carefully controlled and slowly increased until the articular processes are disengaged. The spine is then extended and the traction is reduced. A plaster cuirasse is applied and in many cases the reduction is quite stable. If the injury is a fracture dislocation, and particularly if there is a fracture of one or both articular processes, there may be a danger of redisplacement despite plaster immobilisation.³ In such a case continuous traction must be employed for some weeks before plaster is applied. When there is paraplegia the patient may be too ill for the immediate application of plaster, and continuous traction should again be relied upon.

Until recent years the only available method of applying continuous traction to the neck was by a Glisson's sling which takes its purchase from beneath the chin and the occiput. This method is extremely uncomfortable and it may be responsible for receding of the lower jaw which sometimes remains as a permanent disability and disfigurement. Skeletal traction is free from these complications.

Skeletal traction by skull callipers—Skeletal traction from the skull may be maintained by heavy stainless steel wire, fixed through double burr holes by one of the various types of skull callipers, or by the Roger Anderson calliper which is fixed beneath the zygoma on each side.^{4,5} A suitable type of skull calliper is shown in Figs 530-531. The callipers can be introduced under local anaesthesia without moving the patient from his bed. A 1 to 1½ in. incision is made on each side of the skull above the temporal lines and immediately above the auricles. A small hole is cut with a trephine, through the outer table of the skull. Penetration of the points of the calliper can be controlled by a flange on a screw thread, adjusted according to the thickness of the bone. The points are hooked under the bone margin and the

¹ Walton	New Method of Reducing Cervical Dislocations	<i>Jour. Nerve and Mental Dis.</i> 1893 xx 609
² Walton	Further Observations on Cervical Dislocation	<i>Boston Med. and Surg. Jour.</i> 1903 149 445
³ Soto-Hail	Recurrence of Dislocation of Cervical Spine	<i>Jour. Bone and Joint Surg.</i> 1935 xvi 902
⁴ Cruikshank	Skeletal Skull Traction	<i>South Surg.</i> 1933 li 156
⁵ McKenzie	Fracture dislocation of the spine	<i>Canad. Med. Assoc. Jour.</i> 1933 xxxii 23
⁶ Hoar	Skeletal Traction for Cervical Fracture-dislocation	<i>Arch. Neurol. and Psychiat.</i> 1936 xxxvi 155
⁷ Barton	Cervical Fracture-dislocation—Skeletal Traction	<i>Surg. Gyn. Obst.</i> 1934 lxxv 91

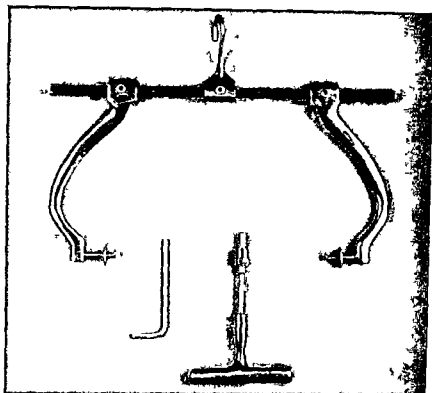


FIG 530

Skull callipers for skeletal traction of the cervical spine with locking key and small trephine



FIG 531

Skull traction using the callipers shown in Fig 530. Within a few hours the cervical dislocation is corrected and the weights are reduced. (In this case the callipers have been inserted too far forwards. The calliper points should lie in a vertical plane passing through the centre of the external ears, where the bone is thicker and there is less risk of perforating the inner table of the skull.)

between the two tables. The limbs of the collipers are then locked and attached to a cord which passes over a pulley at the head of the bed to a weight. The head of the bed is raised on blocks. The pulley should be free to slide from side to side of the crossbar in order to facilitate movement and nursing of the patient. As a rule traction should begin with a weight



FIG 532



FIG 533



FIG 534



FIG 535



FIG 536



FIG 537

Dislocation of interarticular and intervertebral joints (Fig 532). Skull traction was applied (Fig 533). When the overriding of articular processes was corrected (Fig 534) the pulley was lowered to extend the spine and the weight was then reduced (Figs 535-537). After a few weeks the traction was discontinued and plaster applied.

of about 15 or 20 lbs. Check X rays are taken every fifteen minutes until the articular processes are disengaged (Figs 532-537). The crossbar carrying the pulley is then lowered so that the cervical spine is extended. When radiographs show that the articular processes are in accurate apposition the weight may be reduced to 10 lbs. Traction is continued for three or four weeks until the general condition of the patient permits the application of a plaster cast. If the articular processes are fractured and the reduction is unstable traction may be continued for six or eight weeks.

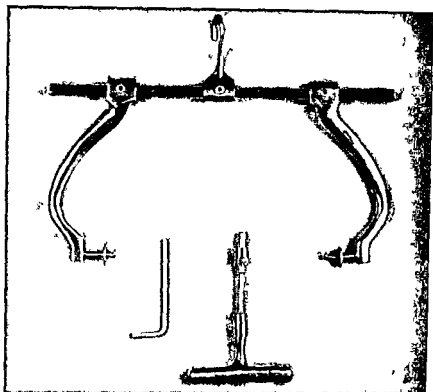


FIG 530

Skull callipers for skeletal traction of the cervical spine, with locking key and small trephine



FIG 531

Skull traction using the callipers shown in Fig 530. Within a few hours the cervical dislocation is corrected and the weights are reduced. (In this case the callipers have been inserted too far forwards. The calliper points should be in a vertical plane passing through the centre of the external ears, where the bone is thicker and there is less risk of perforating the inner table of the skull.)

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FIG 532



FIG 533



FIG 534



FIG 535



FIG 536



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Case of two months' old cervical dislocation with Brown-Séquard paralysis
*—Reduction by skull traction and facetectomy—*A farmer aged thirty five, in attempting to stop a runaway horse, fell beneath the wheels of the cart, and at once complained of pain in the neck, numbness and tingling of both arms, and loss of power in the legs. He was carried to a hospital where radiographs showed a fracture of the sixth cervical spinous process. The vertebræ below this level were concealed by the shadow of the shoulder



FIG. 538

Two months' old unreduced cervical dislocation with interlocked articular processes

A plaster collar was applied but tingling persisted. After several weeks head traction was arranged by a Glisson's sling below the chin. Two months after the accident, radiographs at the Liverpool Royal Infirmary showed a dislocation between the sixth and seventh cervical vertebræ with overriding of the articular processes (Fig. 538). Callus formation beneath the anterior ligament was already fusing the vertebræ in their displaced position. Loss of sensation in the right lower limb, abdomen and lower chest and exaggeration of the reflexes of the left leg with ankle clonus and loss of power, were evidence of injury to the left side of the cord (Brown Sequard syndrome).

Skull clippers were applied with 20 lbs weight extension. The neurological signs were kept under close observation. X-ray examination was repeated daily, and the traction was slowly increased (Figs. 540-547). On the fifth day, when the traction amounted to 40 lbs, the articular processes

slipped into position on the left side. On the right the two processes appeared to be firmly fused despite an oblique pull concentrated on that side they could not be replaced. Distraction of the vertebral bodies had already reached the limit of safety so that on the seventh day facetectomy was performed. The laminae were exposed by a posterior midline incision and the upper half of the right superior articular process of the seventh vertebra was excised. Radiographs taken immediately after operation showed accurate replacement (Fig 547) and the traction was then reduced to 12 lbs. After four weeks the callipers were removed. Immobilisation



FIG 539

Reduction of dislocation after heavy skull tract on for 8 x days
and facetectomy on one side

was continued for two months by a plaster collar and jacket. The sensory loss on the right side slowly receded and ultimately recovered but exaggeration of the reflexes persisted in the left leg.

It is of interest to note the minor discomfort which is associated with heavy skull traction. On the day that the weight was increased to 40 lbs the patient raised his hand mirror cheerfully inspected the callipers in his skull and observed. Anyone looking at this might think it would hurt but it is just nothing to the strap (referring to the Gh son chin sling). That very day his wife presented him with a son and I received a personal honour which I had always understood was reserved for gynaecologists. The son was christened Watson.



FIG. 540

Two months' old unreduced dislocation



FIG. 541

First day—20 lbs. skull traction

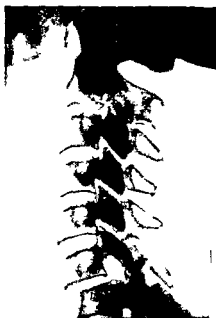


FIG. 542

Second day—20 lbs. skull traction



FIG. 543

Third day—30 lbs. skull traction

Reduction of old cervical dislocation by skull traction and facetectomy



FIG 544

Fourth day—35 lbs skull tract on



FIG 545

Fifth day—40 lbs skull tract on.



FIG 546

Sixth day—10 lbs skull tract on with oblique pull

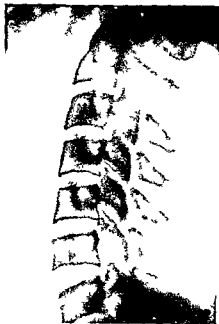


FIG 547

Seventh day—after facetectomy—12 lbs. skull tract on.

Reduction of old cervical dislocation by skull traction and facetectomy

FRACTURES AND DISLOCATIONS OF THE ATLAS

Fracture of the atlas—The usual cause of fracture of the atlas is a fall on the head from a height. Force is transmitted from the skull through the two lateral masses of the atlas which are forced apart so that the bone fractures at one of the weak points represented by the anterior or more commonly the posterior arch (Figs 548-549). There is no cord lesion in at least 50 per cent of cases and the injury is by no means necessarily fatal.

Clinical features—There is spasm of the neck muscles and the patient holds himself rigidly as if balancing a weight upon his head. When getting up from recumbency or with any other change of position the patient usually supports his head with his hands. Nodding and rotation movements are limited. There may be pain and sometimes anæsthesia in the area supplied by the great occipital nerve.

Treatment—If there is paraplegia skeletal

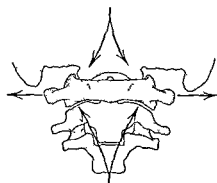


FIG 548

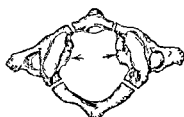


FIG 549

Fracture of the atlas due to a fall on the head. The force is transmitted through the lateral masses which are driven apart so that the arch fractures at its weakest point (After Jefferson)

skull traction should be used for several weeks. If there is no complication the neck is immobilised in plaster with the head in the mid position for three months.

Dislocation of the atlas—The stability of the atlas depends on the transverse ligament which lies between the lateral masses and braces the odontoid process to its anterior arch. If the ligament is torn the atlas can dislocate forwards; similarly if the base of the odontoid process is fractured the atlas and the odontoid fragment may be displaced together. Dislocation is more serious than fracture dislocation because if the odontoid is intact the spinal cord is in danger of being crushed against it (Figs 550-551). When there is less marked displacement and the cord escapes injury the clinical picture resembles that of fracture of the atlas but as a rule there is also rotatory displacement of the head. The bone must be replaced; care being taken to protect the spinal cord by preventing flexion or rotation movement of the patient's head during induction of anæsthesia. A plaster cast is applied with the neck fully extended and the head in neutral rotation; the cast fulving a purchase across the front of the forehead and also being moulded under the chin. If in succeeding weeks it becomes loose enough to permit rotation movements a new cast is applied. Immobilisation is continued for three

months. It is then safe to begin active exercises and within a few weeks to encourage more energetic rehabilitation by which to restore the patient's confidence in his recovery (Chapter XXVII)

Unreduced dislocation of the atlas—If a dislocation of the atlas is overlooked considerable displacement may be observed months or years after injury. Sometimes a perilous degree of displacement becomes firmly stabilised by scar tissue, but in other cases the transverse ligament is weak and attenuated, displacement is progressive, and paraplegia may develop from late compression of the cord. If there is radiographic evidence of increasing deformity or neurological evidence of pressure on the cord, spinal fusion must be undertaken. The spinous processes and laminae of the first three cervical vertebrae are exposed and freshened by the Hibbs' technique. Grafts are inlaid and fixed to the occipital bone as described by Cone and



FIG 550

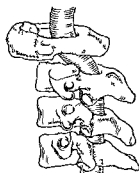


FIG 551

Forward dislocation (Fig 550) and fracture dislocation (Fig 551) of atlas. If the odontoid is displaced forwards there is less danger of cord compression and the patient often survives.

Turner¹. The exposure of this region is difficult, hæmorrhage is not easily controlled, and the operation is one of considerable magnitude.

Spontaneous dislocation of the atlas—A girl aged sixteen, was suffering from tuberculous cervical glands with secondary infection. One day a nurse was dressing the sinuses and in order to gain better access she asked the patient to turn her head. She assisted her by pushing her chin, the girl choked, became cyanosed and was dead². This was an example of spontaneous dislocation of the atlas, the result of decalcification of bone in response to neighbouring infection causing loosening of the attachment of the transverse ligament (see Fig 421 p 263).

Spontaneous subluxation of the atlas—Spontaneous dislocation of the atlas and sudden death is rare, but spontaneous subluxation with "acquired torticollis" is more frequent^{3,4}. It nearly always occurs in children, aged six to twelve years and it may complicate any infection in the upper part of the neck. Many cases have occurred after tonsillitis and some after

¹ Cone and Turner. Treatment of Cervical Fracture Dislocations by Skeletal Traction and Fusion. *Jour Bone and Joint Surg.* 1937, xiv, 844.

² Reid quoted by Craig. Clinical Observations on the Surgical Pathology of Bone. Edinburgh 1931.

³ Watson Jones. Spontaneous Hyperextension Dislocation of the Atlas. *Lancet* 1903, xxv, section of Orthopedics 58.

⁴ Watson Jones and R. F. Roberts. Calcification, Decalcification and Ossification. (review of reported cases of spontaneous dislocation of the atlas). *Brit Jour Surg.* 1931, xxi, 476.



FIG 552

Hyperextension fracture dislocation of the atlas and odontoid. Inset shows A.P. view through open mouth. Compare with normal below.



FIG 553

The dislocation was reduced but the patient was elderly and plaster could not be tolerated. A block leather 'doll collar' was used (Reduced by Mr. A. H. H. at the Robert Jones and Agnes Hunt Orthopaedic Hospital).

nasopharyngitis retropharyngeal abscess tuberculous adenitis acute mastoid infection and osteomyelitis of the occipital bone About a week after the onset of infection the child complains of a crick in the neck The head is held rigidly in the position of torticollis but there is no contracture or spasm of the sternomastoid the rigidity and deformity are due to spasm of the deep cervical muscles Radiographs show forward and rotatory displacement of the atlas (Fig 420) The displacement must be reduced under anaesthesia Plaster is applied with the head in neutral rotation and extension Recumbency skull traction and chin traction are unnecessary After the primary focus of infection is healed immobilisation is continued for ten weeks A removable collar may be used for two or three weeks longer until a full range of painless movement is regained

Flexion fracture dislocation of atlas and odontoid—It is often said that the gravity of dislocation of the atlas is increased if there is also a fracture of the odontoid process¹ Precisely the opposite is the case If there is a fracture of the odontoid process the chances of survival are increased and with suitable treatment the patient makes a complete recovery there should be no permanent disability The atlas and the odontoid process are displaced together as a rule there is both forward and rotatory displacement Under anaesthesia the head is turned to the neutral position the cervical spine is extended and a closely moulded plaster is applied Immobilisation is continued for three months If reduction of the displacement is accurate the odontoid fracture usually unites by bone



Fig 551

Hyperextension fracture dislocation of the atlas and axis

Hyperextension fracture-dislocation of atlas and odontoid—Sometimes the displacement is in the opposite direction and the atlas together with the odontoid process is displaced backwards Such a displacement is shown in Fig 552 The antero posterior radiograph taken through the open mouth shows the fracture of the base of the odontoid process and slight lateral displacement of the interarticular joints The displacement was reduced by flexion of the head and a plaster jacket was applied but the patient was elderly and querulous and within a few days it was necessary to replace the plaster with a moulded block leather 'doll collar' (Fig 553)

Hyperextension fracture dislocation of atlas and axis—In some cases the line of injury passes between the second and third cervical vertebral bodies and through the weak part of the neural arches of both vertebrae (Fig 554) The bodies of both atlas and axis are tilted backwards Occasionally the spinal cord is contused or extradural haemorrhage causes paralysis and death If the spinal cord escapes injury the treatment is the same as in hyperextension fracture dislocation of the atlas

¹ I have recently published text books state that a dislocation of the atlas with fracture of the odontoid process is as a rule fatal This of course is quite untrue

CHAPTER XVII

VERTEBRAL FRACTURES AND DISLOCATIONS WITH PARAPLEGIA

Injuries of the spinal cord and cauda equina may be classified into two groups—

- 1 Paraplegia due to loss of conductivity from contusion compression or traction injury without destruction of nerve cells or nerve fibres. These lesions are capable of complete recovery, whether sustained by the cauda equina or by the spinal cord.
- 2 Paraplegia due to more severe injury with disruption of nerve cell connections and laceration or crushing of the nerve fibres. Only the nerves of the cauda equina can recover after such an injury. In the spinal cord itself destructive damage cannot be repaired for regeneration of the nerve fibres is impossible.

The clinical picture during the first two or three weeks may be the same whether the lesion of the cord is a block to conductivity or an actual loss of continuity. In each case there is inhibition of cord activity below the lesion with flaccid motor paralysis, absence of reflexes, loss of superficial and deep sensation, trophic skin disturbances and visceral paralysis with retention of urine. If the paralysis and sensory loss are not total the inhibition is clearly incomplete and the lesion is more likely to be a simple recoverable block than an irrecoverable crushing injury. If paralysis is total the lesion may be either a simple block or a crushing injury and not until a few weeks have elapsed is it possible to judge with certainty whether the lesion is recoverable or not. In many cases however the probabilities can be assessed by radiographic examination and repeated clinical examination.

RADIOGRAPHIC EXAMINATION

The severity of the nerve injury varies with the type of fracture and with the level of lesion. Radiographic evidence may make it possible to decide whether transection of the cord is probable, possible or unlikely. It must be emphasised however that the degree of displacement shown in the radiograph is not necessarily that which existed at the time of injury (p. 304). Inferences must be drawn from the potentialities of displacement disclosed by the radiograph rather than from the position of the vertebra at any one moment.

Fractures of the sacrum.—Nerve root injury is rare because the foramina are more than twice the size of the nerve roots which emerge through them. When there is gross forward displacement of the lower fragment of the

sacrum all the distal sacral nerves may be crushed and there is incontinence and gluteal anaesthesia

Wedge compression fracture—In simple wedge fractures there is no displacement of the interarticular or intervertebral joints. Paralysis is rare and if it does arise the lesion is almost certainly due to simple spinal concussion. Complete recovery is usually observed within a few days or weeks.

Comminuted hyperflexion fracture—The acute flexion injury which sharply angulates two vertebrae on each other and produces a comminuted fracture with intervertebral disc injury is more likely to cause momentary contusion of the cord. In the rare event of a posterior marginal fragment being forced backwards there may be actual compression of the cord or cauda equina either at the time of injury or during attempted reduction. If such a fracture is left untreated the lumen of the canal may be progressively reduced by increasing deformity and callus formation and even if there has been no primary nerve injury delayed paraplegia may supervene after many months. If the pressure is promptly relieved the prognosis in these cases is usually good.

Lumbar fracture-dislocation—Fracture dislocation of the spine is the bone injury most commonly associated with paraplegia. The nerve fibres are contused, compressed or severed between the upper posterior margin of the lower vertebral body and the laminae of the displaced vertebra above. In the lumbar region the lumen of the spinal canal is wide and the nerves of the cauda equina are relatively mobile. Displacement even of gross degree may cause no more than simple nerve compression. Moreover the nerves are capable of regeneration. In fracture dislocations below the first lumbar level which are reduced and immobilised recovery from paraplegia may be expected in at least two thirds of the cases.

Lumbar fracture dislocation with locking of the articular processes deserves special attention. If the spine is hyperextended without first unlocking the processes recovery from paraplegia is practically impossible because the nerve injury is increased by attenuation. On the other hand it has now been proved that complete recovery is possible after early operative reduction (p. 325).

Dorsal fracture-dislocation—In the upper dorsal region the cord almost completely fills the neural canal and its mobility is restricted by the anchorage of nerve roots and the ligamentum denticulatum. Forward displacement of the upper segment of the spine of more than minimal degree must crush the cord (Fig. 555). Severe fracture dislocations in this region are almost invariably associated with complete transection and an irrecoverable paraplegia. In the lower half of the dorsal region the cord injury may be less severe and a few cases have recovered after postural reduction of the displacement (Figs. 556, 557). The narrowness of the escape in these cases has been proved by persistent exaggeration of deep reflexes or by a permanent Babinski response to plantar stimulation.

Cervical dislocation—In the cervical region when the ligaments of the interarticular and intervertebral joints are torn there is free mobility of the dislocated segment of the spine. The most severe degrees of primary displacement may be masked by complete spontaneous reduction. In some cases there is no radiographic evidence of bone injury at all and yet the cord is irrecoverably damaged. Ascending haemorrhage and oedema

may rapidly prove fatal. The prognosis must therefore be guarded. On the other hand, even complete dislocations with paraplegia may be associated with less severe cord injury, and if compression is relieved by manipulative reduction or by skeletal traction recovery is possible. In fractures and fracture dislocations of the atlas paraplegia is often incomplete and recovery is possible. Delayed paraplegia from recurrent displacement and late cord compression is sometimes observed.

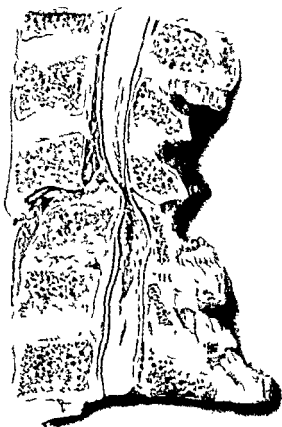


FIG. 505

Specimen of fracture dislocation of spine showing crushing of cord between the upper angle of the vertebral body below and the laminae of the vertebra above. There is also pressure on the cord by backwardly displaced disc material. (Liverpool Univ. by Pathological Museum)

Fracture of the laminae—In very exceptional cases fragments of the laminae or spinous processes are forced into the spinal canal, causing direct contusion and compression of the cord and necessitating early laminectomy.

CLINICAL EXAMINATION

Clinical features^{1, 2}—Clinical examination shortly after the accident shows complete flaccid paraplegia with absence of superficial and deep reflexes, loss of skin and deep sensation and incontinence of feces and retention of

¹ Riddoch "Spinal Injuries" *Proc. Roy. Soc. Med.* 1927, xxi, 63.

² Naffziger "Neurological Aspects of Injuries to the Spine" *Jour. Bone and Joint Surg.* 1933, xx, 444.

urine with overflow incontinence. The upper limit of anæsthesia is often marked by a zone of hyperæsthesia at the actual level of cord injury.

Decubitus ulceration.—During the first few weeks there is a great danger of sloughing of the skin and subcutaneous tissues at points of pressure, particularly over the sacrum, the trochanters and the heels. This is partly due to the enforced immobility of the paralysed patient to the loss of sensation of tissues which are subjected to pressure and to soiling and infection of the skin from urinary and rectal incontinence. It is also due to trophic skin changes which are most severe during the first few weeks. If the patient survives even although there is permanent motor and sensory paralysis, the tendency to decubitus ulceration gradually becomes less pronounced.

Bladder infection.—There is retention of urine with overflow incontinence and a susceptibility to bladder infection. This like the skin ulceration is due to motor sensory and trophic disturbances. The motor paralysis accounts for poor emptying stagnation and fermentation of urine. The loss of sensory innervation predisposes to instrumental injury to the mucous membrane of the urethra and bladder. Trophic changes impair the physiological powers of defence and account for vasodilatation or even hæmorrhage into the submucous tissues.

Cerebro spinal fluid.—Examination of the cerebro spinal fluid may be of value in proving subdural hæmorrhage which usually indicates a grave intraspinal lesion. Sometimes, in cervical dislocations there is evidence of spinal block in the lack of response of the fluid pressure to jugular vein compression (Queckenstedt's test). This may disappear after reduction of the dislocation by skeletal traction. As a rule, however, examination of the fluid is of no value. In lumbar, dorsal and some cervical injuries although at the moment of injury the displacement may have been such as to produce spinal block the laying of the patient on a flat surface reduces the deformity sufficiently to restore communication of the fluid above and below the level of injury. A normal pressure response does not therefore exclude the possibility of gross displacement at the moment of injury nor does it exclude the possibility of reproducing such displacement if the spine is acutely flexed.

Incomplete paralysis.—The distinction between a complete and an incomplete lesion of the cord can be made by a simple clinical test. If one of the patient's toes is flexed and compressed in such a way that if his sensation were normal severe pain would be induced, and if the patient perceives no sensation and shows no reaction whatever, the absence of pain and of deep sensation may be accepted as evidence of complete sensory loss. If the patient recognises some sensation from the test, the lesion of the cord is incomplete. Every degree of incomplete lesion may be observed from the paralysis which disappears within twenty-four hours, to the one which slowly improves over a period of many weeks or even months and in which some degree of residual paralysis and alteration of reflexes remains permanently. In incomplete cauda equina lesions improvement may still be expected two years or even longer after injury, and even in injuries of the cord, incomplete lesions may continue to improve for a surprisingly long time.

Complete spinal cord paralysis.—If an injury to the cord is characterised by complete and total interruption and this evidence persists for more

than a few days, it is almost certain that the cord is transected. Certainly after three weeks, complete failure to respond to painful deep sensation in the toes indicates an irrecoverable lesion. The stage of total inhibition and flaccid paralysis persists for about three weeks. Reflex function of the cord which is cut off from the higher centres is then established. Stimulation of the sole of the foot gives rise to reflex with drawal from the stimulus. Gradually the field of reception of afferent stimuli increases and the response of efferent motor stimuli extends. Ultimately the flaccid paraplegia passes into spastic paraplegia, stimulation of any part of the lower limbs accounts for a mass reflex, and the power of the spastic calf, hamstring and adductor muscles produces the usual contractures. As reflex activity appears in the lower cord, reflex control of the bladder is established. At frequent intervals, but without the knowledge or control of the patient, the sphincters are relaxed, the detrusor musculature contracts, and the bladder is emptied by a powerful urinary stream as broad and continuous as that of normal micturition.¹

Complete cauda equina paralysis—Irrecoverable lesions of the cauda equina are characterised by paralysis of the peripheral type. The muscles remain flaccid and there is no recovery of superficial or deep sensation. The tendency to trophic skin ulceration becomes less marked and a type of bladder control returns. The bladder is still cut off from the cortical and thalamic centres so that normal conscious control is impossible, and it is cut off from the sacral spinal segments so that reflex control is impossible. The only nerve control remaining is that initiated by the peripheral nerve ganglia and this is very imperfect. The sphincters are almost if not entirely inefficient, but contraction of the detrusor musculature of the bladder wall is also inefficient.² Without the reinforcement of voluntary abdominal wall muscles, the detrusor musculature may be incapable of overcoming the mechanical resistance of the elastic tissues surrounding the urethra in the region of the triangular ligament. This resistance takes the place of the normally innervated sphincters and the abdominal muscles take the place of the normally innervated detrusor musculature. Considerable straining efforts are necessary, the stream is thin and it is interrupted whenever the patient pauses for a breath, but relative comfort is possible, for apart from the accidents which accompany coughing and sneezing the victim can remain dry. If therefore he can succeed in overcoming retention and emptying the bladder by straining he is in a much happier condition than the patient with a transverse cord lesion who though he has a perfect reflex mechanism, is unable to control it.

TREATMENT OF VERTEBRAL FRACTURES WITH PARAPLEGIA

First-aid treatment—*The victim of a spinal injury complicated by paraplegia must never be lifted by the shoulders and hips in such a way that the spine sags between the points of support.*³ If the patient is lying face upwards and is lifted in this manner, forcible flexion of the lumbo-dorsal spine momentarily increases the displacement and may inflict serious damage on the cord (Fig. 355). The face down position is relatively safe for the spine is then

¹ Denny Brown and Graeme Robertson *Brain* 1933 lvi 149-39.

² Watkins *The Bladder Function in Spinal Injury* *Brit. Jour. Surg.* 1936, xxiii, 724.

³ Watson Jones *Fractures of Spine* First Aid Treatment *Brit. Med. Jour.*, February 1931 i 209, November 1933 ii 940.

hyperextended, and in most spinal fractures hyperextension reduces the displacement and relieves cord compression. We now know, however, that hyperextension is not invariably safe, for there are a few rare spinal injuries in which even this position may increase the nerve injury (Chapter XV). The main object of the first aid worker must be to keep the spine straight. As soon as possible a flat stretcher or board is placed alongside the patient, who is carefully rolled on to it. If he is lifted at all this must be done by many slings of bandage or other material passed under the dorso lumbar junction as well as under the head, shoulders and hips. Once on the stretcher the patient must not be moved from it until radiographic examination has disclosed the exact nature of the injury.

The original teaching of ambulance workers to turn a patient with a spinal injury on to his back before lifting him by the shoulders and hips, was obviously wrong for it produced a dangerous degree of flexion of the spine. But the most recent suggestion of a special first aid stretcher which fully hyperextends the spine even before the victim leaves the site of accident¹ is a swing to the opposite extreme and is also wrong. The ambulance worker must aim at the neutral position. Hyperextension must be performed only by the surgeon after complete radiographic investigation has proved that it is necessary and safe.

Should the fracture be reduced?—The principle of early reduction of uncomplicated spinal fractures is now accepted, but the application of this principle to the fracture with paraplegia is not generally accepted. It is easy to argue in favour of immediate reduction and immobilisation because this obviously relieves nerve compression and prevents further injury. The cord and spinal nerves have been contused and are possibly compressed by displaced bone. Replacement of the bone in its normal position by manipulation is clearly more rational than the removal of bone by laminectomy (Figs 556 557).

The objection lies in the fear that the plaster used to maintain the reduction, being applied over insensitive tissues, will increase the dangers of decubitus ulceration². The fact remains however, that there is no alternative treatment which can be relied upon. Hyperextended beds and frames, and slings under the patient passed to overhead beams are just as certain to be complicated in this way and the use of water beds is even more likely to produce pressure sores. It is also argued that the cord injury was determined at the moment of accident by the severity of the original blow, and that subsequent compression is unimportant, and indeed is non-existent if the patient lies flat in bed³. This argument overlooks the demands of nursing. The patient must be continually twisted and turned and lifted, and these manoeuvres are easily capable of reproducing the original displacement. We must conclude that the best treatment for the cord injury is reduction and immobilisation of the fracture at the earliest possible moment.

Lumbar fractures and dislocations : plaster jacket—In the lumbar region the prognosis is so good, and it is so probable that the paraplegia will recover within a few weeks that as a rule there can be no serious objection to the usual routine treatment—postural reduction by hyperextension and

¹ Padula Spinal Hyperextension Litter *Jour Bone and Jo nt Surg* 1932 xx 507

² Irwin Fractures of the Spine *Brit. Med Jour* January 1936 I, 1

³ Jefferson Spinal Injuries *Proc Roy Soc Med.*, 192 xxi 62.

immobilisation in a plaster jacket. The patient is turned on to his right side, his face his left side and his back at hourly intervals. Every effort is made to keep him dry and if necessary windows may be cut in the plaster to reduce the pressure over prominent bones. A new jacket is applied as soon as recovery from the nerve lesion permits it. In a few cases of lumbar injury with paraplegia so complete that early recovery is unlikely



FIG. 336

Fracture dislocation of eighth lumbar vertebra with spinal cord injury producing almost complete paraplegia

the treatment which is used for dorsal injuries with complete paraplegia may be adopted.

Dorsal fractures and dislocations plaster bed—In dorsal injuries where the prognosis is more doubtful and where there is a greater likelihood of complete and prolonged paralysis a plaster bed is used, sometimes with skeletal traction. The fracture is reduced by laying the patient face downwards on a firm table with the spine extended by pillows and sandbags under the shoulders and pelvis. Leg suspension by the Davis technique may be added if the patient's general condition permits it. A plaster bed is made by applying many plaster slabs over the patient's back until there is a uniform thickness of from $\frac{1}{2}$ to 1 in. Quick setting plaster must be used. The bed is at once removed, dried by hot air and well padded with thick felt or sorbo rubber. Meanwhile the skin of the back is thoroughly cleaned and alcohol

and camphor are applied. The plaster bed is then replaced and the patient is turned.

Immediate steps must be taken to relieve pressure over the sacrum and heels. This is most easily accomplished by using skeletal traction from both tibial tubercles¹. Two Brauns splints are used with 10 or 15 lbs of extension and the foot of the bed is raised. Adhesive strapping traction



Fig 5o7

Same case as Fig 5o8 after manipulative reduction (radiograph through plaster). The paraplegia fully recovered.

to the forefoot keeps the heels off the splints and prevents drop foot. Subsequently an anterior plaster lid or turning case may be constructed so that the patient can be turned without moving the spine. If the paraplegia recovers within a few weeks an ordinary plaster jacket is applied. If the nerve lesion remains complete and is obviously irrecoverable the immobilisation and skeletal traction are discontinued after eight or ten weeks.

Cervical fractures and dislocations. **skull traction**—In cases of complete paraplegia at the cervical level the patient is usually desperately ill, the chest muscles are paralysed and respiration is entirely diaphragmatic. The most that can be done is to apply skull callipers under local anaesthesia and to defer the application of a plaster jacket until the general condition is improved and there is sign of recovery from the paralysis.

¹ Döller. *Treatment of Fractures*. Bristol 4th ed. 1935. 146.

Indications for laminectomy—The only certain indication for laminectomy in recent injuries is the exceptionally rare case of depressed fracture of the laminae or spinous processes. In lumbar injuries with cruda equina paralysis which fail to show early recovery laminectomy may be justifiable for we are dealing with peripheral nerves which are capable of regeneration.^{1,2} Late operative treatment is usually hopeless because the nerve roots are so extensively matted.

In dorsal and cervical injuries of the cord there is some doubt as to whether laminectomy is ever of value. In the only cases where the operation can do more than is possible by simple reduction of the displacement—cases of ascending paralysis due to spreading hæmorrhage and œdema—the patient is usually too ill to permit of any interference at all.

In the later stages laminectomy may be indicated for the delayed paraplegia of untreated comminuted fractures involving the neural canal and of cervical fractures and dislocations with recurrent displacement.

Care of the bladder—Urinary infection is the usual cause of death after fracture of the spine with paraplegia. Of 339 spinal bladder cases reported by Thomson Walker³ in 1917 nearly 50 per cent died of urinary sepsis within two months and the ultimate death roll from ascending urinary infection and uræmia was estimated to be 80 per cent. The factors which predispose to infection of the paralysed bladder have already been discussed (p. 351) the exciting cause is catheterisation. Intermittent catheterisation is disastrous no matter what precautions are adopted infection is almost certain to supervene. Even an indwelling catheter with intermittent washing of the bladder may be unsafe during the first few weeks when there are pronounced trophic changes in the mucosa of the urethra and bladder. Thomson Walker reported that cases treated in this way arrived in hospital with part of the urethral floor and overlying structures sloughed out at the peno scrotal angle leaving a gap of 1 to 2 in. as the result of combined urethritis and the pressure of a tied in catheter. It is true that a tied in catheter with tidal drainage^{4,5} and an antiseptic fluid seal may be successful in well-equipped hospitals under skilled supervision but as an emergency measure the technique is unsafe. The management of the paralysed bladder must be referred at the earliest possible moment to a skilled urological surgeon⁶ and meanwhile the orthopædic surgeon should be guided by the following principles—

(1) *Retention of urine must be relieved promptly*—For the same reason that pressure over the sacrum causes trophic skin ulceration and bedsores pressure due to distension of the bladder causes trophic mucosal ulceration and cystitis. Retention of urine must be relieved despite the fact that the patient complains of no pain and does not demand relief.

(2) *No catheter should be passed*—The orthopædic surgeon should not accept the risk of passing a urethral catheter. When management of the bladder has been handed over to a skilled urologist it is possible that he may elect to use an indwelling catheter and tidal drainage but meanwhile retention must be relieved by other methods.

¹ Oldberg, Neuro-surgical Considerations of Fractures of Spine. *Surg Clin of America* 1936 xvi 921.

² Naftziger, Neurological Aspects of Spinal Injury. *Jour Bone and Joint Surg* 1935 xx 445.

³ J. Thomson Walker, *Ian et 191* 1 13 *J. of Roy Soc Med* 191 xxx 1931.

⁴ H. S. Lawrie and E. W. Nathan, Antiseptic Tidal Drainage of the Bladder. *Ian et 1919* ii 10.

⁵ D. Munro, *Journal of Urology* 1936 xxxvi 1.

⁶ G. E. Ward, Management of the Bladder in Spinal Injuries. *Reports of Med in Warfare* cited by His Majesty's Stationery Office, London, 1941.

(3) *Distension of the bladder with overflow is unsafe*—Despite reports from one clinic that if no single passage of a catheter is permitted the bladder may be allowed to distend and overflow,^{1 2} the general consensus of opinion is that this method is unsafe because intravesical pressure causes trophic ulceration

(4) *Manual expression of the bladder is unsafe*—The technique of emptying the paralysed bladder by manual expression is unsafe because there is risk of rupturing the bladder

(5) *Aspiration of the bladder is relatively safe*—The pubis is shaved the skin sterilised and a lumbar puncture needle inserted in a backward and downward direction urine is drawn off with a 20 cc aspirating syringe The aspiration must be repeated at six or eight hourly intervals If this method is continued too long it causes pre vesical cellulitis

(6) *Suprapubic cystotomy is safe*—As soon as the patient is admitted to hospital suprapubic cystotomy may be performed If the lesion is above the eleventh dorsal segment no anæsthetic is necessary, but if it is below this level general anæsthesia should be used The bladder is exposed freely through a midline incision and picked up securely with tissue forceps 1 in. apart a de Pezzar or Malecot catheter is introduced through an appropriate trocar and cannula or by means of a bladder perforator It is important that the fistula should be made high half way between the symphysis and umbilicus in order to prevent leakage The catheter is connected by sterile rubber tubing to a water seal bottle on the floor beside the bed No irrigation of the bladder is necessary unless there is already infection, and even then the bladder must not be distended

The management of irrecoverable paraplegia—The normal and healthy individual finds it difficult to believe that a paraplegic patient can overcome his anguish enjoy comparative freedom from pain, achieve complacency, engage in sedentary occupations and games, become mentally normal and finally lead a happy life But disabled sailors soldiers airmen and civilians have proved that this can be true³ The important points in nursing are

(1) to prevent infection of the urinary tract which is the cause of death in nearly all paraplegics (2) to prevent trophic ulceration and bed sores, (3) to mitigate the discomforts of incontinence, (4) to ensure adequate sleep

Drainage of the bladder is necessary for the control of urinary infection As a rule the bladder must be washed out daily, either suprapubically, or by means of a urethral catheter through the bladder and out by the suprapubic tube The suprapubic tube must be changed at regular intervals, preferably each day When the patient is up in his wheel chair or hand-propelled tricycle the catheter is connected with a rubber urinal suspended from the wrist strapped to the thigh, and emptied by a tap near the ankle If there is no suprapubic drainage a urinal with a penile sheath is used For the control of rectal incontinence it is wise to spend one, or perhaps two days a week in bed—“enema days” when the colon is emptied by high irrigation with water soap and turpentine With such a routine, social

¹ Connor and Nash Urological Complications of Injuries of the Spine (fifty-four non-catheterised cases treated by distension and overflow without a single urinary tract infection) *Amer Jour Surg* 1931 xxxi 103

² D Kidd *Brit Med Jour* April 1919 i 39

³ F L Cowland The After-treatment of Paralytic Patients following Injuries to the Spine (report after seventeen years' experience as Consultant of the Star and Carter House for Paraplegics) *Brit Med Jour* 1941 i 814

engagements are possible on the other days of the week without fear of incontinence. Unremitting nursing care of the whole body is necessary to avoid trophic ulceration of the skin. The patient should sleep on a

Dunlopillo mattress. his wheel chair must be well padded with cushions. the sacrum, hips and lower extremities must be constantly watched suitably cleaned, hardened with spirit and powdered with talc. Two or three baths a week are valuable. if the bath is at right angles to the wall and two orderlies are available the problem is facilitated. It is sometimes possible with the aid of crutches and surgical splints of the caliper type to teach patients to walk with a tripod gait. Little imagination is needed to realise the sense of achievement and tremendous psychological stimulus of even the most halting progression.

CHAPTER XVIII

LOW BACK STRAIN AND SCIATICA

"No problem has been more perplexing than the low back pain of orthopedics, but with increased knowledge and after an immense amount of research, order and hope have evolved from chaos"—WALTER MERCER

In the lumbo sacral region there is a complex anatomical system of more than twenty joints with innumerable ligaments, muscles and aponeuroses. These joints are as susceptible to trauma as the joints of the limbs, they may suffer ligamentous injury, muscle strain, cartilage displacement, subluxation, dislocation or traumatic arthritis. But whereas the knee joint, for example, is a single and easily accessible joint, the lumbo sacral system of joints lies deeply, and the various components cannot be examined individually. Difficulties arise from the closely related nerve supply. The posterior muscles, fasciæ and ligaments of the lumbar interarticular and intervertebral joints and the sacro-iliac and lumbo sacral joints are all supplied by the posterior primary divisions of the lumbar nerves. Pain arising in any of these structures may be referred as a reflex phenomenon throughout the distribution of the low lumbar nerves to the back of the thigh, the leg and the foot. Steindler has proved that sciatica may arise reflexly in any low back strain, even although there is no direct compression of the nerve.¹ In addition to this source of referred pain, there may also be actual compression and irritation of the sciatic nerve or its roots. This may occur within the spinal canal where the cauda equina lies close to the intervertebral joints, in the intervertebral foramina where the nerve roots are in contact with the interarticular joints, or in any part of the peripheral course of the nerve trunk through the muscles and aponeuroses of the lumbar and gluteal regions.

Syndrome of "Sciatic Scoliosis"²—Whether the pain arises reflexly or by direct compression, and whether its source is within the spinal canal, in the intervertebral foramen or in the peripheral course, the radiation into the thigh and leg is the same. Furthermore, whether the symptoms in the low back are due to a painful muscle, a painful ligament or joint or an irritated nerve, the same muscle guard is observed, and there is lumbar rigidity, an unwillingness to flex the spine, and sometimes a list to one side producing slight asymmetry of the trunk so that the curve of one loin is more pronounced than the other. In more severe cases there is obvious deformity described as "total scoliosis" (Fig. 558). Similarly, straight

¹ Steindler and Luck. *Jour Amer Med Assoc* 1933, ex 106

² Watson Jones. *Sciatica and Sciatic Scoliosis*. *The Practitioner*, 1937, cxxxix 473

leg raising not only stretches the sciatic nerve but by tightening the hamstring muscles and tilting the pelvis it strains the sacro iliac joints flattens the lumbar spine and applies tension to all the posterior ligaments muscles and aponeuroses. Limitation of this movement from the normal range of 80° to 30° or 40° (Lasegue sign) may be observed in any of the injuries which cause low back pain and sciatica. All these symptoms and signs—lumbar and gluteal pain, sciatica, lumbar rigidity, total scoliosis and limitation of straight leg raising—make up the syndrome of sciatic scoliosis which may arise from many pathological lesions.

Classification of low back strains—

The lesions responsible for sciatic scoliosis can be classified into three groups—

Myofascial and ligamentous injuries

- 1 Strain of sacro spinalis or of gluteal muscles especially at their periotendal attachments
- 2 Fascial contractures and adhesions following strain
- 3 Strain of sacro iliac or lumbosacral ligaments and of interspinous ligaments with or without spinous process impingement

Joint injuries

- 1 Strain or subluxation of interarticular sacro iliac and lumbosacral joints
- 2 Strain of previously arthritic joints

Intervertebral disc injuries

- 1 Rupture of posterior common ligament with retro-pulsion of intervertebral disc into the spinal canal or intervertebral foramen
- 2 Fibrosis and hypertrophy of ligaments flava

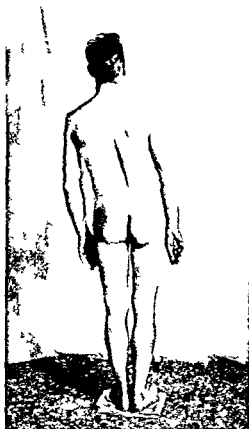


FIG. 308

Typical sciatic scoliosis with list of spine to left. This may be due to fascial ligamentous articular or intervertebral disc injury.

Etiology—The responsible injury may immediately precede the onset of symptoms or there may be an interval of some months. The accident is sometimes a fall from a height in the standing or sitting position or a flexion force such as is capable of producing a vertebral body fracture. More often it is a strain sustained by lifting a heavy weight. The lumbar region of man has been modified to meet the requirements of the erect posture and its structural efficiency is greatest in that position. In the stooping position the muscles and ligaments are on stretch and the joints bear a cross strain. This susceptibility to injury is often increased by the architectural deficiencies which are so common at the lumbosacral

junction¹ Horizontal sacrum spondylolysis spina bifida, posterior displacement of the fifth lumbar vertebra sacralisation of transverse processes, asymmetrical articular facets and impingement of spinous processes are not so much the primary causes of pain in the back as factors predisposing to injury^{2,5}

MYOFASCIAL INJURIES

Sacrospinalis and gluteal strain—The common sites of myofascial strain are (1) the origin of the sacrospinalis from the back of the sacrum between the spinous processes and the posterior superior iliac spine and (2) the origin of the gluteus maximus from the posterior superior iliac spine and the sacro tuberos ligament

Clinical features—In addition to the classical features of sciatic scoliosis—lumbar pain sometimes sciatica lumbar rigidity especially to flexion movement a list of the trunk to one side and limitation of straight leg raising—there is tenderness on pressure over the periosteal attachment of the injured muscle There may also be diffuse tenderness and hyperæsthesia over the whole of the muscle sheath The absence of a history of previous recurrent attacks of low back pain excludes many types of sciatic scoliosis The differentiation from other acute injuries depends entirely upon the localisation of tenderness This localisation should be accurately confirmed by the following test

Steindler's novocaine test⁶—The tender point is marked and the skin is anaesthetised with a few drops of novocaine The needle is inserted deeply and the involved tissue is identified by the unusually sharp pain which is elicited when the point of the needle comes in contact with it From five to ten cubic centimetres of 1 per cent novocaine are injected Five postulates must be met before the diagnosis is proved (1) contact of the needle aggravates the local pain (2) contact of the needle elicits or aggravates the painful radiation into the thigh and leg (3) novocaine injection suppresses the local pain (4) novocaine injection suppresses the radiating pain (5) novocaine injection restores normal straight leg raising movement

Treatment—The object of treatment is the repair of torn tissues without extensive adhesion formation or fascial contracture Early forcible passive stretching and manipulation increase the fibrous exudation and aggravate scar tissue formation On the other hand scar contracture is encouraged by undue delay in the practice of active exercises During the early acute stage protection by firm bandaging a lumbar belt or even a plaster jacket is necessary Radiant heat and diathermy are contraindicated they increase the local hyperæmia swelling and exudation When heat is combined with deep massage the pain is usually aggravated and muscle spasm is increased If electrotherapy and physiotherapy are used at all the treatment must be confined to light soothing massage When the acute symptoms have subsided active exercises are practised and in the later stages physiotherapy

¹ Frank Dickson Low Back Injuries with Reference to Patology of Congenital Abnormalities (review of 99 cases) *Southern Med Jour* 1936 xxix 364

² Lee Lumbo-sacral Pains for Low Back Pain *Jour Bone and Joint Surg* 1936 xlii 33

³ Danforth and P. W. Sisson Anatomy of Lumbo-sacral Region in Relation to Sciatic Pain *Jour Bone and Joint Surg* 1937 xlii 199

⁴ Cockitt and Variations in the Anatomic Structure of the Lumbar Spine *Jour Orthop Surg* 1939 416

⁵ Johnson and Smith Posterior Luxation of Lumbo-sacral Joint *Jour Bone and Joint Surg* 1931 xvi 867 and 87

⁶ Steindler Differential Diagnosis of Pain Low in the Back *Jour Amer Med Assoc*, 1934 cx, 106

and electrotherapy may be beneficial. The possibility that the injury has been no more than an exciting cause lighting up a toxic fibrositis must be borne in mind. Colonic stasis and distant foci of infection should be excluded.¹

Repeated novocaine injection of Leriche—In some cases the actual tissue injury is of minimal degree and yet the disability is prolonged and severe fascial contracture may arise. Leriche believes that these changes are due to stimulation of the sensory nerve endings in the injured tissues. Afferent impulses pass to the cord, efferent autonomic impulses arise and

the vessels in the neighbourhood of the injury are dilated (Fig. 39).

Vaso dilatation causes effusion and the outpouring of metabolites further stimulates the nerve endings and maintains the reflex stimulation. A vicious circle is set up and the fibrinous exudation is out of all proportion to the degree of local injury. If the reflex arc is cut by novocaine injection the circle is broken and recovery is accelerated. Novocaine injection is important therefore not only as a diagnostic procedure but also as a therapeutic measure.² It may be repeated every third or fourth day. The patient should be warned of the temporary reaction of pain which may be very severe after several hours when the analgesia passes off.

Fascial contractures following strain—Myofascial adhesions and contracture may supervene. The symptoms of low back pain and

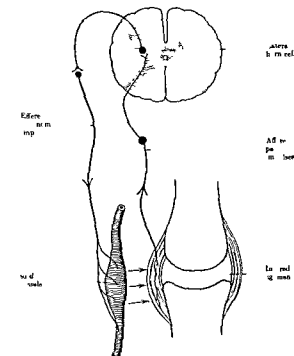


Fig. 39

Leriche's theory of the reflex source of persistent exudation after simple sprains which may explain the value of repeated novocaine injection.

sciatica persist with recurrent acute exacerbations. The contracture is most marked in the gluteal fascia below the posterior superior spine or anteriorly in the region of the tensor fasciae latae and the ilio-tibial band. It sometimes produces an actual abduction contracture of the hip which may be demonstrated by Ober's test.³ The patient lies on his sound side with the hips sufficiently flexed to obliterate the lumbar lordosis. The upper leg is flexed to a right angle at the knee. The ankle is lightly grasped with one hand and the hip is steadied with the other. The limb is then abducted and extended at the hip until the thigh is in line with the trunk. If there is an abduction contracture the limb remains passively abducted by the shortened ilio-tibial band which can be felt tightly stretched.

¹ Albee, *Myofascitis*, *Amer. Jour. Surg.* 1934, xlviii, 5.

² Campbell, *On the Injuries of the Lower Limbs*, *Jour. Roy. Naval Med. Serv.* Jan. 1932.

³ Ober, *Role of the Ilio-tibial Band and the Tensor Fasciae Latae in Low Back Lesions*, *Ann. N.Y. Acad. Sci.* 1936, xlviii, 105.

Ober's operation—An incision is made from the anterior superior spine to the trochanter. The ilio tibial band and fascia lata are incised and the intermuscular septa are divided. The cut surfaces are sometimes of almost cartilaginous hardness and they retract 2 in. or more as soon as the incision is complete. Relief usually begins within a week of operation, straight leg raising steadily increases and the low back pain disappears within two or three months.

Stripping of the posterior superior spine—If in the acute stage the symptoms were localised by clinical and novocaine tests to the region of the posterior superior spine, the muscles and fasciæ should be stripped from the iliac crest exactly as in Smith Petersen's exposure of the sacro iliac joint.¹ It was the almost immediate relief of sciatica by Smith Petersen's exposure of the ilium for suspected bone or joint lesions which led Roberts² and Heyman³ to recognise the therapeutic possibilities of this simple division of soft tissues.

Manipulation of the spine—If the initial symptoms were related to the sacro spinals and its aponeurosis rather than to the gluteal muscles and fasciæ, and if there is no abduction contracture of the hip, the spine may be manipulated under anaesthesia. The decision to manipulate a spine for sciatic scoliosis must be made however only after careful consideration. Some surgeons have reported many successful cases,⁴ but on the whole the manipulative treatment of sciatic scoliosis has been disappointing. Very frequently after temporary relief the disability recurs. It is always difficult to be sure that there is no factor other than simple adhesion formation, and if the symptoms are due to the displacement of an intervertebral disc manipulation of the spine is dangerous and may even produce paraplegia.

Strain of sacro-iliac and interspinous ligaments—The strain may be concentrated not on the spinal and gluteal muscles, but on the ligaments of the sacro iliac joints and the interspinous and supraspinous ligaments. Localisation may be possible by clinical examination and by the novocaine injection test. Treatment is the same as for myofascial strains. Repeated novocaine injection by the technique of Leriche is of value, and in the later stages manipulative treatment may be necessary.

Excision of spinous process—If a lesion of an interspinous ligament resists conservative treatment the possibility of aggravation by impingement of adjacent spinous processes should be considered. Removal of one of the impinging processes has sometimes given relief.

JOINT INJURIES

The strain may have involved the sacro iliac, lumbo sacral or inter-articular joints, and in each case the sciatic scoliosis syndrome develops. Deep tenderness may be elicited over the involved joint, and localisation is sometimes possible by deep exploration with a needle and by novocaine injection. A long history of recurrent attacks persisting despite physio-

¹ Smith Petersen. *Jour. Bone and Joint Surg.* 1926 viii 112.

² Roberts quoted by Ober. *Jour. Bone and Joint Surg.* 1936 xviii 10.

³ Heyman. Relief of Sciatic Pain. *Jour. Bone and Joint Surg.* 1934 xvi 801.

⁴ Bankart. *Brit. Med. Jour.* 1936 ii 416.

therapeutic treatment with no evidence of fascial contracture is suggestive of joint involvement. Careful radiographic examination is of importance for in many cases the injury is only an exciting factor lighting up a pre existing arthritis.

Sacro-iliac arthritis—The surgeon must be continually on guard to exclude *sacro iliac tuberculosis* which often develops insidiously apparently as the result of injury and which may simulate the traumatic types of sciatic scoliosis. Sooner or later, radiographic examination will disclose the typical bone destruction. Symptoms related to the sacro iliac joint developing after parturition are often due to low grade *infective arthritis*. The infection may never advance to the stage of abscess formation and yet symptoms persist until the pelvis and lumbar spine are immobilised in plaster.

Spondylitis deformans (ankylosing spondylitis) is often lighted up by injury. It gives rise to the typical sciatic scoliosis syndrome and in the early stages may be erroneously diagnosed as simple low back strain. Radiographic evidence of new bone formation round the intervertebral joints appears only after the symptoms have been present for several years. There are two distinguishing features. The rigidity involves not only the lumbar spine but the whole spine to the cervico dorsal junction and lateral and hyperextension movements are limited as well as flexion movement. The whole spine moves in one piece. Secondly many months or years before radiographic changes appear in the spine there is sclerosis of bone round both sacro iliac joints. This is the first stage of the sacro iliac arthritis which progresses to complete disappearance of the joint space and ultimately to bony ankylosis. This is the one type of arthritis which responds dramatically to radiotherapy. Complete relief from pain can be assured and by suitable hyperextension exercises and the temporary use of a posterior spinal support deformity can be controlled.

Osteo-arthritis of the lumbar spine is shown radiographically by the multiple osteophytes at the margins of the vertebral bodies and by narrowing of the intervertebral joint spaces. Radiographs taken in the oblique plane may disclose arthritic changes in the interarticular joints.¹ It is important to recall that many middle aged individuals show osteophyte formation round the lumbar vertebral bodies and that this is not necessarily a source of pain. The surgeon should continue in his search for other causes of low back pain and sciatica.

Lumbo-sacral arthritis—Narrowing of the lumbo sacral intervertebral disc space has been noted in some cases of sciatic scoliosis by many observers. The degenerative and traumatic arthritis which results in narrowing of the disc may be a direct cause of low back pain and reflex sciatica. Furthermore sciatic pain may be the result of nerve root compression from two possible sequelae. As the disc disappears the adjacent vertebral bodies approximate their articular processes subluxate and there is encroachment on the intervertebral foramen. The nerve root pressure may demand facetectomy.² Secondly if the disc is not merely degenerated but actually displaced backwards

Putti Anatomia dell'Artrite Vertebrale Apofisario (Ist. ortopedico Fazzoli Bologna 1933, 1902, 1903, 1904, 1905, 1906, 1907, 1908, 1909, 1910, 1911, 1912, 1913, 1914, 1915, 1916, 1917, 1918, 1919, 1920, 1921, 1922, 1923, 1924, 1925, 1926, 1927, 1928, 1929, 1930, 1931, 1932, 1933, 1934, 1935, 1936, 1937, 1938, 1939, 1940, 1941, 1942, 1943, 1944, 1945, 1946, 1947, 1948, 1949, 1950, 1951, 1952, 1953, 1954, 1955, 1956, 1957, 1958, 1959, 1960, 1961, 1962, 1963, 1964, 1965, 1966, 1967, 1968, 1969, 1970, 1971, 1972, 1973, 1974, 1975, 1976, 1977, 1978, 1979, 1980, 1981, 1982, 1983, 1984, 1985, 1986, 1987, 1988, 1989, 1990, 1991, 1992, 1993, 1994, 1995, 1996, 1997, 1998, 1999, 2000, 2001, 2002, 2003, 2004, 2005, 2006, 2007, 2008, 2009, 2010, 2011, 2012, 2013, 2014, 2015, 2016, 2017, 2018, 2019, 2020, 2021, 2022, 2023, 2024, 2025, 2026, 2027, 2028, 2029, 2030, 2031, 2032, 2033, 2034, 2035, 2036, 2037, 2038, 2039, 2040, 2041, 2042, 2043, 2044, 2045, 2046, 2047, 2048, 2049, 2050, 2051, 2052, 2053, 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2386, 2387, 2388, 2389, 2390, 2391, 2392, 2393, 2394, 2395, 2396, 2397, 2398, 2399, 2400, 2401, 2402, 2403, 2404, 2405, 2406, 2407, 2408, 2409, 2410, 2411, 2412, 2413, 2414, 2415, 2416, 2417, 2418, 2419, 2420, 2421, 2422, 2423, 2424, 2425, 2426, 2427, 2428, 2429, 2430, 2431, 2432, 2433, 2434, 2435, 2436, 2437, 2438, 2439, 2440, 2441, 2442, 2443, 2444, 2445, 2446, 2447, 2448, 2449, 2450, 2451, 2452, 2453, 2454, 2455, 2456, 2457, 2458, 2459, 2460, 2461, 2462, 2463, 2464, 2465, 2466, 2467, 2468, 2469, 2470, 2471, 2472, 2473, 2474, 2475, 2476, 2477, 2478, 2479, 2480, 2481, 2482, 2483, 2484, 2485, 2486, 2487, 2488, 2489, 2490, 2491, 2492, 2493, 2494, 2495, 2496, 2497, 2498, 2499, 2500, 2501, 2502, 2503, 2504, 2505, 2506, 2507, 2508, 2509, 2510, 2511, 2512, 2513, 2514, 2515, 2516, 2517, 2518, 2519, 2520, 2521, 2522, 2523, 2524, 2525, 2526, 2527, 2528, 2529, 2530, 2531, 2532, 2533, 2534, 2535, 2536, 2537, 2538, 2539, 2540, 2541, 2542, 2543, 2544, 2545, 2546, 2547, 2548, 2549, 2550, 2551, 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3382, 3383, 3384, 3385, 3386, 3387, 3388, 3389, 3390, 3391, 3392, 3393, 3394, 3395, 3396, 3397, 3398, 3399, 3400, 3401, 3402, 3403, 3404, 3405, 3406, 3407, 3408, 3409, 3410, 3411, 3412, 3413, 3414, 3415, 3416, 3417, 3418, 3419, 3420, 3421, 3422, 3423, 3424, 3425, 3426, 3427, 3428, 3429, 3430, 3431, 3432, 3433, 3434, 3435, 3436, 3437, 3438, 3439, 3440, 3441, 3442, 3443, 3444, 3445, 3446, 3447, 3448, 3449, 3450, 3451, 3452, 3453, 3454, 3455, 3456, 3457, 3458, 3459, 3460, 3461, 3462, 3463, 3464, 3465, 3466, 3467, 3468, 3469, 3470, 3471, 3472, 3473, 3474, 3475, 3476, 3477, 3478, 3479, 3480, 3481, 3482, 3483, 3484, 3485, 3486, 3487, 3488, 3489, 3490, 3491, 3492, 3493, 3494, 3495, 3496, 3497, 3498, 3499, 3500, 3501, 3502, 3503, 3504, 3505, 3506, 3507, 3508, 3509, 3510, 3511, 3512, 3513, 3514, 3515, 3516, 3517, 3518, 3519, 3520, 3521, 3522, 3523, 3524, 3525, 3526, 3527, 3528, 3529, 3530, 3531, 3532, 3533, 3534, 3535, 3536, 3537, 3538, 3539, 3540, 3541, 3542, 3543, 3544, 3545, 3546, 3547, 3548, 3549, 3550, 3551, 3552, 3553, 3554, 3555, 3556, 3557, 3558, 3559, 3560, 3561, 3562, 3563, 3564, 3565, 3566, 3567, 3568, 3569, 3570, 3571, 3572, 3573, 3574, 3575, 3576, 3577, 3578, 3579, 3580, 3581, 3582, 3583, 3584, 3585, 3586, 3587, 3588, 3589, 3590, 3591, 3592, 3593, 3594, 3595, 3596, 3597, 3598, 3599, 3600, 3601, 3602, 3603, 3604, 3605, 3606, 3607, 3608, 3609, 3610, 3611, 3612, 3613, 3614, 3615, 3616, 3617, 3618, 3619, 3620, 3621, 3622, 3623, 3624, 3625, 3626, 3627, 3628, 3629, 3630, 3631, 3632, 3633, 3634, 3635, 3636, 3637, 3638, 3639, 3640, 3641, 3642, 3643, 3644, 3645, 3646, 3647, 3648, 3649, 3650, 3651, 3652, 3653, 3654, 3655, 3656, 3657, 3658, 3659, 3660, 3661, 3662, 3663, 3664, 3665, 3666, 3667, 3668, 3669, 3670, 3671, 3672, 3673, 3674, 3675, 3676, 3677, 3678, 3679, 3680, 3681, 3682, 3683, 3684, 3685, 3686, 3687, 3688, 3689, 3690, 3691, 3692, 3693, 3694, 3695, 3696, 3697, 3698, 3699, 3700, 3701, 3702, 3703, 3704, 3705, 3706, 3707, 3708, 3709, 3710, 3711, 3712, 3713, 3714, 3715, 3716, 3717, 3718, 3719, 3720, 3721, 3722, 3723, 3724, 3725, 3726, 3727, 3728, 3729, 3730, 3731, 3732, 3733, 3734, 3735, 3736, 3737, 3738, 3739, 3740, 3741, 3742, 3743, 3744, 3745, 3746, 3747, 3748, 3749, 3750, 3751, 3752, 3753, 3754, 3755, 3756, 3757, 3758, 3759, 3760, 3761, 3762, 3763, 3764, 3765, 3766, 3767, 3768, 3769, 3770, 3771, 3772, 3773, 3774, 3775, 3776, 3777, 3778, 3779, 3780, 3781, 3782, 3783, 3784, 3785, 3786, 3787, 3788, 3789, 3790, 3791, 3792, 3793, 3794, 3795, 3796, 3797, 3798, 3799, 3800, 3801, 3802, 3803,

into the spinal canal or intervertebral foramen the displaced nodule exerts pressure on the nerve roots of the cauda equina.

Interarticular arthritis—In other cases there is no clinical or radiographic evidence of sacro iliac lumbosacral or intervertebral arthritis but the myofascial type of sciatic scoliosis has been excluded and neurological investigation may have excluded pressure within the spinal canal. There is sometimes evidence of distant foci of infection of recurrent boils of a rheumatic tendency or of mild rheumatoid changes. The presumption is that strain has lighted up an interarticular arthritis. It is difficult to confirm this radiographically or clinically but the frequency of arthritic changes in these joints is proved by post mortem examinations. The relief which follows plaster immobilisation helps to confirm the presumption.

Treatment—In nearly all cases where the sciatic scoliosis syndrome persists and recurs relief may be secured by immobilisation in a plaster jacket applied with slight head traction (Fig 560). The traction enlarges the intervertebral foramina and there may be immediate relief from agonising pain as soon as the jacket is applied. The subsequent treatment depends on the type of case and two groups must be differentiated (a) where an underlying arthritis is proved and (b) where a simple traumatic arthritis is presumed.

Cases of proved arthritis—If there is sclerosis of bone adjacent to the sacro iliac joints and diffuse rigidity indicating a general spondylitis the plaster jacket is retained for two or three months until pain is completely relieved and it is then replaced by a celluloid jacket or posterior spinal support which must be worn for several years (Fig 561). When fixation of all interarticular and intervertebral joints is so sound that pain cannot recur the support is discarded gradually. If there is sacro iliac arthritis which is not part of a general spondylitis the joint should be arthrodesed by the Smith Petersen technique (Fig 572). If there is lumbosacral arthritis a lumbosacral arthrodesis is indicated¹ but only after the possibility of retropulsion of the intervertebral disc has been excluded. The fusion may be combined with excision



FIG 560

Application of head traction plaster jacket for sciatic scoliosis

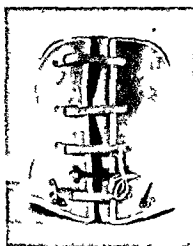


FIG 561

Celluloid jacket for more permanent immobilisation in recurrent sciatic scoliosis

¹ Compare Operative Treatment for Low Back Pain (294 patients with low back pain—67 in operations) *Journal of the American Medical Association* 1934 xiv 749

of the articular processes if intervertebral foramen pressure is suspected

Cases of presumed traumatic arthritis—Where no underlying arthritis has been proved the plaster jacket is retained until there has been complete freedom from pain for at least one month. As a rule this involves plaster fixation for not less than three months. The plaster is then discarded. A simple corset or sacro iliac belt may be worn for a short period. The patient is allowed to recover movements of the spine by his own cautious activity. He is kept under continued observation. If an acute attack recurs in subsequent months neurological investigation must be undertaken to exclude the third type of sciatic scoliosis—that due to retropulsion of an intervertebral disc. Only if this is excluded are we justified in assuming that there is a recurrent arthritis which must be treated by the continued protection of a celluloid jacket for many months or even years.

INTERVERTEBRAL DISC INJURIES

In 1896 Kocher¹ reported the case of a man who fell 100 ft. in the standing position. Autopsy revealed a rupture of one of the lumbar intervertebral discs without fracture of the vertebral body. In 1911 Middleton² operated on the spine of a man who lifted a heavy weight felt a snap in the back and within a few hours developed flaccid paraplegia. A firm white mass was removed which resembled the central pulp of the intervertebral disc. In the same year Goldthwait³ reported a case of manipulation of the spine under anaesthesia to reduce a subluxated sacro iliac joint which was followed immediately by paraplegia. He concluded that backward displacement of the lumbo sacral intervertebral disc was responsible for this catastrophe and probably also for other cases of lumbago and sciatica.

These three cases are the first records of retropulsion of the nucleus pulposus of an intervertebral disc and they illustrate the three types of injury which may produce or aggravate such a displacement—falls from a height flexion strain while lifting a heavy weight and manipulation of the spine under anaesthesia. More recently cases have been described of retropulsion following puncture of a disc by a lumbar puncture needle which had been pushed too far across the spinal canal⁴.

Pathological features—It is probable that degenerative changes in the annulus fibrosus and posterior longitudinal ligament may weaken the peripheral part of a disc and predispose to injury⁵. Nevertheless the traumatic factor is definite. The displacement usually occurs in healthy young adults between the ages of thirty and forty. In 50 per cent of cases there is a history of injury immediately preceding the onset of symptoms and in a further 30 per cent a history of injury followed by a latent period. The lesion is three times as common in men as in women and although any level

Kocher De Verletzungen des Wirbelsäule Zugfall als Beitrag zur Pathologie der Muskulatur
Rückenmarks Mit a d Gren geb d Med u Chir 1896 1 415
Middleton and Teher Injury of Spinal Cord due to Rupture of Intervertebral Disc during Muscular Effort *Chicago Med Jour* 1911 lxxvi 1
Goldthwait Lumbo-sacral Articulation. Examination of Some Cases of Lumbago Sciatica and Paraplegia. *Boston Med and Surg Jour* 1911 cx 205
Pace Injuries to the Vertebra and Intervertebral Discs following Lumbar Puncture. *Amer Jour Dis Child* 1935 xlix 849
Joplin The Intervertebral Disc. *Surg Gyn Obst* 193 lxix 591

of the spine may be involved the large majority involve the lumbo sacral disc (30 per cent) or the disc between the fourth and fifth lumbar vertebrae (20 per cent) ¹

The intervertebral discs form an anterior wall to the spinal canal exactly at the level where the nerve roots are passing laterally to emerge through the intervertebral foramina. Small midline protrusions may therefore arise without nerve pressure and may be found in routine post mortem examinations. On the other hand a lateral displacement occurs at a point where the nerve root cannot escape friction and pressure (Fig 36). It is the attempt to avoid this frictional and compression neuritis which accounts for lumbar rigidity and total scoliosis. The list may be towards or away from the side of pain depending on the anatomical relationship of the mass to the nerve root. As a rule only one nerve root is involved. The overlap of areas supplied with sensation by the lumbar and sacral nerves is so complete that it is impossible to refer pain in one region of the limb to a particular nerve root. For the same reason sensory impairment or anaesthesia is seldom demonstrated because as Foerster has shown at least two sensory nerve roots must be severed before there is even a limited area of anaesthesia in the limb ². It is therefore impossible to prove nerve root compression or to localise the level of the lesion by clinical tests. Special radiographic investigation is necessary.

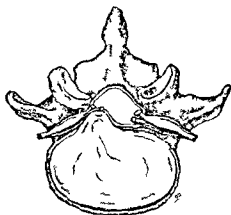


FIG 36

Retropulsion of an intervertebral disc into the spinal canal or intervertebral foramina causes nerve root friction and compression

Clinical features—The clinical picture is that of sciatic scoliosis and there is no symptom or sign which is pathognomonic. A vigorous young man is lifting a weight feels a snap in his back and immediately complains of pain in the lumbo sacral region. There is lumbar rigidity, loss of the normal lumbar lordosis and probably a list of the spine to one side. At some time thereafter sciatica develops. The pain is very severe, it is aggravated by sneezing and coughing and it may be almost impossible to turn in bed. There is sometimes tingling and numbness in the foot and toes. Straight leg raising is limited to 90° or 30° on the painful side and somewhat limited on the opposite side. If the nerve root irritation is of long standing the ankle jerk is absent but there is usually no other neurological sign.

The clinical features so far described are characteristic of any variety of sciatic scoliosis. The type of injury and the immobilising intensity of the pain may lead the surgeon to suspect displacement of a disc but the symptoms could also be due to myofascial ligamentous or articular injury. The injection of lipiodol for X-ray screening which is necessary to prove displacement of the disc involves some risk of inducing an irritative leptomeningitis, it is not an investigation to be undertaken lightly. At

¹ J. S. Barr: Relationship of Intervertebral Disc to Back Strain. *Surgery St. Louis* 1933 iv 1
² Foerster: The Dermatomes in Man. *Brain* 1933 1 11

this stage therefore the patient is usually treated by immobilisation in a head traction plaster jacket. Friction of the nerve root is controlled, pain is relieved and within two or three months it may disappear. Sometimes treatment by osteopathy or by manipulation under anaesthesia has been pursued and there may have been sudden and dramatic relief from the pain. There is little doubt that displacement of the fibro cartilage may to some extent be reduced for the hole in the annulus fibrosus is large enough to permit movement of the fragments of cartilage in each direction. Sooner or later however the disc material is redisplaced and the mass within the spinal canal becomes bigger. Nerve friction gives place to nerve compression and each attack of pain is likely to be more severe and less capable of relief by immobilisation in plaster. Ultimately the agonising pain can be controlled only by heavy doses of morphine and the victim may even be driven to drug addiction.

If the intraspinal mass is of sufficient size more than one nerve root may be involved. Areas of anaesthesia develop there may be bilateral sciatica and even paraplegia may supervene. Long before this stage has been reached neurological and radiographic investigation should be undertaken.

Neurological investigation—The mass is seldom of sufficient size to obstruct the spinal canal completely. In most cases therefore the cerebrospinal fluid below the level of lesion shows a normal pressure response to jugular compression (Queckenstedt's test). Chemical analysis shows no evidence of an extremely high protein level of xanthochromia or of spontaneous clotting (syndrome of From). Nevertheless there is usually some stasis of fluid and if it is withdrawn from a low enough level the protein content is often found to be raised above the normal 20 to 30 mg per cent. A content of 50 to 100 mg is suggestive. The protrusion may however be so far to the side of the spinal canal that there is no stasis of cerebrospinal fluid at all and a normal fluid does not therefore exclude the possibility of retropulsion of a disc.

Radiographic investigation—Routine X-ray examination usually shows no abnormality. The fourth lumbar or lumbosacral disc space is sometimes narrowed and taken in conjunction with the clinical history this may be suggestive. However the disc space may be reduced because the disc is degenerated and not because it is displaced. Special radiographic examination to visualise the whole lumbosacral sac is necessary.

Air myelography—It may be possible to prove retropulsion of a disc and to establish the level by air myelography.¹² With the patient lying on his side on an X-ray table which is tilted up 45° at the foot end a spinal puncture needle is inserted at the second lumbar interspace. Spinal fluid and air are exchanged in 5 c.c. volumes until air returns from the needle. As a rule about 30 c.c. of air are necessary to outline the lumbosacral sac in adults. Stereoscopic lateral and antero-posterior radiographs are taken. The patient is subsequently kept in the Trendelenburg position for six hours flat on the back for twelve hours and then gradually elevated to the erect position over a period of forty-eight hours. This procedure reduces the severe headache which otherwise develops. The interpretation of the films is not easy and it is doubtful whether a negative result can be accepted as conclusive.

*Indications for lipiodol myelography*¹⁰—The injection of iodised oils into the subarachnoid space is to be regarded as a dangerous procedure" (Loyal Davis)¹⁰ Subarachnoid injections of iodised oil should be avoided,



FIG 563

Retropulsion of intervertebral disc involving fourth lumbar root causing filling defect in the lipiodol



FIG 564

Retropulsion of the fourth lumbar intervertebral disc causing almost complete obstruction to the flow of lipiodol

at least until all other means of diagnosis have been exhausted (American Council of Pharmacy)¹¹ The dangers of the irritant effect of lipiodol are so serious that it should not be used until the diagnosis has been established on clinical grounds and a decision has been made to operate moreover the oil must be removed at operation *If the history and clinical signs suggest the probability of retropulsion of a disc routine radiographic and clinical examinations have excluded other causes the case has resisted conservative treatment for more than six months and the symptoms have recurred after the temporary relief of three months immobilisation in plaster operative exploration is indicated and the injection of lipiodol may then be justified as an immediate pre operative measure*

*Lipiodol myelography*¹²—Four cubic centimetres of warm oil are injected through a wide bore spinal puncture needle at the second or third lumbar interspace The patient should be screened at once A tilting X ray table is essential and the spine is screened with the patient lying prone Large

¹ Alajouanine et Petit-Dutail lls. *Presse méd.* 1930, xxxviii 1657 and 1 49

² Galland *Arch. Franco-Belges de Chir.* 1930 xxxii 479

³ Galland *Bull. et Mém. Soc. de Méd. de Paris* 1930 ii, 58

⁴ Mixer and Barr *New England Jour. Med.* 1934 cccii 10

⁵ Mixer and Ayer *New England Jour. Med.* 1935, cccxiii 389

⁶ Barr *Jour. Bone and Joint Surg.* 1937 xix 3 3

⁷ Glorieux *La Hernie Postérieure du Ménisque Intervertébrale* Paris 1937

⁸ Williams *Jour. Bone and Joint Surg.* 1937, xix 343

⁹ Love, Crafon and Camp *Jour. Bone and Joint Surg.* 1937 xix 7 6.

¹⁰ Davis Ha en and Stone *Jour. Amer. Med. Assoc.* 1930 xciv 772.

¹¹ Report Council on Pharmacy and Chemistry *Jour. Amer. Med. Assoc.* 1932, xcix 1946.

¹² Hampton and Robinson *Röntgenographic Demonstration of Rupture of Intervertebral Disc after Injection* *Amer. Jour. Roent.* 1936 xxxvi 82.

masses of displaced disc material cause an almost complete obstruction to the flow of lipiodol which only squeezes past drop by drop (Fig 564). Smaller masses give rise to a localised filling defect and the nerve root which should be emerging at that level fails to fill (Fig 563). Since the mass usually occupies one side of the canal and the lipiodol flows freely down the other side lateral films seldom show any filling defect. Sometimes a protrusion may be so small that even when screened in the antero-posterior plane the filling defect is seen only as the oil flows towards or away from that level like an island washed by the tide it is the first to be uncovered and the last to be submerged.

Conservative treatment—It is possible to control the root pain associated with small lesions by the continued protection of a plaster or celluloid jacket. The protection must be continued for many years or even permanently.

Operative treatment—The spinal canal is exposed by removal of the laminae of the vertebrae above and below the affected disc. It is possible to remove the displaced cartilage by an extra-dural approach but if lipiodol has been used the dura should be opened for its removal. The projection into the spinal canal is always smaller than the filling defect shown in the radiographs. Sometimes it seems disappointingly small but the rapid and complete relief which follows excision proves that it has caused frictional neuritis though not necessarily compression neuritis. The dura of the anterior wall is incised over the mass. The displaced fragments of cartilage may pop out like loose bodies out of a knee joint but more often they require traction and dissection. Fragments of tissue looking like rolled up wet blotting paper are removed. The sensory root emerging at the level of the lesion should usually be excised. This causes little or no disability it relieves post-operative pain and it prevents recurrent symptoms due to fibrosis and scarring. The lipiodol is extracted by a suction apparatus and the incisions in the dura are closed with fine sutures. The vertebrae above and below should be fused by laying a bone graft and bone chips subperiosteally across the gap in the laminae. The wound is closed and drained for twenty-four hours. There is sometimes retention of urine which may require catheterisation for a few days. In most cases recovery is complete within two or three months.

Hypertrophy of ligamenta flava²⁶—Similar clinical symptoms and signs and the radiographic evidence of a filling defect in the column of lipiodol, may be due to fibrosis and hypertrophy of the ligamenta flava. The relationship of this pathological condition to displacement of the discs and to trauma in general is not yet known. The condition no doubt explains some of the cases in which an intervertebral disc was believed to be ruptured, operation failed to disclose any displacement, and yet the laminectomy and removal of the ligament relieved the symptoms.

- R. Jaeger "Intervertebral Disc Injury Its Relation to Intervertebral Neuralgia" *Rocky Mount Med Jour* 1938 xxxv 205
 * Felsberg "Observations upon Sixty Laminectomies" *Surg Gyn Obst* 19 x i 11
 * Towne and Rechart "Compensation of Lumbo-sacral Roots by Thickened Ligamenta Flava" *Ann of Surg.* 1931 xciv 897
 Spurling and Mayfield "Hypertrophy of Ligamenta Flava as a Cause of Low Back Pain" *Jour Amer Med Assoc* 1937 cix 998
 * Naffziger, Inman and Saunders "Lesions of the Intervertebral Disc and Ligamenta Flava" *Surg Gyn. Obst* 1938, lxxvi 22
 * Brown "Enlargement of Ligamentum Flavum." *Jour Bone and Joint Surg* 1938 xx 33

CHAPTER XIX

INJURIES OF THE PELVIS

Fractures and dislocations of the pelvis may be divided into three groups (1) avulsion fractures due to muscular violence (2) fractures and dislocations of the pelvic ring due to crushing injuries and (3) injuries of the sacrum and coccyx

AVULSION FRACTURES OF THE PELVIS

Sudden and uncontrolled effort may detach any of the muscles arising from the pelvis and avulse fragments of bone from the point of origin. The powerful long bellied muscles of the thigh are most commonly involved.

Anterior inferior iliac spine—rectus femoris avulsion—A boy is playing Rugby football his enthusiasm is greater than his strength and he is determined to convert a try however difficult the angle and however distant the posts. At the moment of kicking the ball there is a sharp pain in the groin the player falls to the ground and active flexion of the hip is found to be painful and limited. Radiographs show slight downward displacement of a fragment of bone from the anterior inferior iliac spine just above the margin of the acetabulum. The bone has been avulsed by the rectus femoris muscle (Fig 565). This fracture is to be distinguished from the epiphyseal line of a separate ossicle of bone which may develop normally in this situation. There is no indication for operative suture. Recumbency for a few weeks with the hip flexed to a comfortable position is the only treatment necessary.



FIG 565

Avulsion of anterior inferior iliac spine by rectus femoris sustained by a schoolboy playing Rugby football

Anterior superior iliac spine—sartorius avulsion—Forceful contraction of the sartorius muscle may avulse the bone of the anterior superior iliac spine. The fragment is slightly displaced but again there is no indication for operation. The pain is relieved by flexion of the hip. Displacement may be imperfectly corrected but functional recovery is complete within about two months.

Epiphysis of ischium—hamstring avulsion—The hamstring muscles arise from the tuberosity of the ischium and a bone fragment may be avulsed by sudden muscular contraction especially in youths whose epiphyses are not united. Fig 566 shows avulsion of the epiphysis of the tuber ischii sustained by a track runner during a hundred yards' sprint.¹ The track surface was imperfect, and the injury was sustained at the moment that a slight hollow in the ground called for an increased and unexpected muscular effort. There was complete recovery by conservative treatment, and the epiphysis united firmly with considerable new bone formation.



FIG 566

Avulsion of ischium by hamstrings sustained by a track runner

ISOLATED INJURIES OF THE PELVIC RING

The two innominate bones, by their articulation with the sacrum posteriorly and with each other at the symphysis pubis, form an intact pelvic ring. If a fracture breaks the continuity of this ring at only one level, gross displacement of the fragments cannot arise. If there is a second injury to another part of the ring, considerable displacement of the segment of bone separated by the two fractures may be seen. Isolated injuries of the ring include slight separation of the symphysis pubis, unilateral fracture of one or both pubic rami, fracture of the body of the ilium, and subluxation of the sacro iliac joint (Figs 567-570).

Fractures of the pubic rami—The most common injury is unilateral fracture of one or both pubic rami. Movement of the fragments is limited and displacement is minimal. Complete immobilisation is therefore unnecessary. The only treatment indicated is recumbency for a few weeks. Recovery should be complete within a period of from two to three months.

Isolated fractures of the ilium and minor separations of the symphysis pubis also recover fully without special treatment. The surgeon must, however,

¹Cohen. Avulsion of Ischial Tuberosity (with review of the three previously reported cases two of which were track runners). *Jour Bone and Joint Surg* 1937 xix 1133

satisfy himself that separation of the symphysis pubis is in fact an isolated injury. If there is wide displacement of the pubic bones there is almost certainly a second injury in the sacro iliac region which may easily be overlooked and which often accounts for persistent disability.

Sacro-iliac subluxation is the only isolated pelvic ring injury which is of serious significance. Even minor displacements of this joint may account for persistent pain and incapacity. The injury is to be recognised clinically by the localisation of pain, the tenderness over the joint and the typical displacement. The ilium is pushed slightly backwards and towards the midline. The posterior superior iliac spine is therefore more superficial.

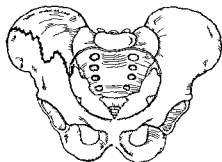


FIG 56



FIG 56b

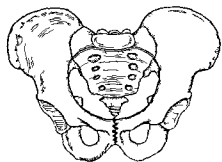


FIG 57

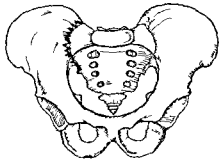


FIG 58

Isolated injuries of the pelvic ring. There is no marked displacement and no special treatment is indicated.

than the corresponding bony prominence of the opposite side and it lies nearer to the spinous processes (Fig. 571). Radiographs also show that this part of the ilium is unduly close to the midline and that it overlaps the shadow of the sacrum to an abnormal degree (Fig. 582).

The displacement is reduced in the same way that complete disruptions of the pelvis are reduced by rotating the ilium forwards (p. 377). The patient lies on the normal side and pressure is applied over the front of the crest of the ilium. The joint is immobilised in plaster for three months. Pain persisting indefinitely points to imperfect reduction to an unstable joint or to traumatic arthritis. Sacro-iliac arthrodesis should be performed by the Smith-Petersen technique¹ (Fig. 572).



FIG 51

Prominence of the posterior part of the ilium indicates sacroiliac subluxation or dislocation. The displacement is easily felt and as in this case on the left side it may be obvious on inspection.

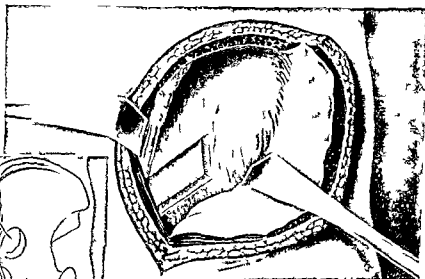


FIG 52

Sacro-iliac arthrodesis

(St. Peter's technique)

Through an incision centered on the posterior superior iliac spine and following the crest of the ilium the dorsum ilii is exposed subperiosteally. A rectangular block of bone is cut out of the ilium exactly over the sacroiliac joint. The block is lifted out and the articular cartilage on its deep surface removed. Cartilage from the articular surface of the sacrum is then excised by a rongeal gouge and finally the block of bone is replaced and punched deeply into the cavity so that it locks into the sacrum.

COMBINED INJURIES OF THE PELVIC RING

The pelvic ring is made up of the anterior pubic segments which are developed for the protection of the pelvic viscera and for the attachment of muscles and the postero lateral iliac segments which also serve the function of weight bearing. Combined fractures of the pelvic ring are of two types. In the first type both fractures lie in the pubic segments. In the second one fracture is in the pubic segment and one in the weight bearing iliac segment.

Combined injuries of pubic segment of pelvic ring—Double fractures of the pubic part of the pelvis are the result of lateral crushing. The victim may be standing sideways to a wall and he is crushed by a motor vehicle which strikes the opposite side of the pelvis. The injury may be a bilateral fracture of both pubic rami or a unilateral fracture of both rami with separation of the symphysis pubis (Figs 573-574). The detached fragment

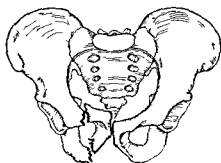


FIG 573

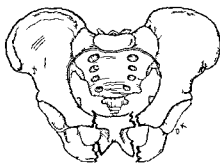


FIG 574

Combined injuries of the pubic segment of the pelvic ring produced by lateral compression of the pelvis. There is only slight displacement. Patients should be nursed on their backs. (*Bycotte's of First Jour Surg for the Am J Surg* 1928 xx 3)

of bone is relatively small. Its displacement is limited by the attachments of many muscles and whatever the degree of displacement there is no shortening of either limb and no alteration in the alignment of weight bearing joints. As a rule therefore it may be ignored. The patient is treated in recumbency for five or six weeks; the lateral compression of the pelvis should not be increased by allowing the patient to lie on one side.

Combined injuries of iliac and pubic segments of pelvic ring—The commonest combined injury causing complete disruption of the pelvis is a dislocation of the symphysis with a dislocation of the sacro iliac joint. Less frequently there is a dislocation of the symphysis with a fracture of the ilium near the sacro iliac joint or a fracture of both pubic rami with a dislocation of the sacro iliac joint (Figs 575-577). One half of the pelvic girdle is widely displaced carrying with it the lower limb so that there is deformity and shortening.

Unlike the first type of combined injury which is produced by lateral compression of the pelvis these injuries are produced by antero posterior compression. The patient is standing with his back to the wall when he

is crushed by a motor vehicle or he is lying on the roadway and the wheel of the vehicle mounts one side of the pelvis. In other cases heard on collisions are responsible. The mechanism of injury is important because it gives the clue to the technique of manipulative reduction.

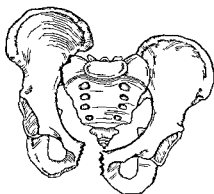


FIG. 55

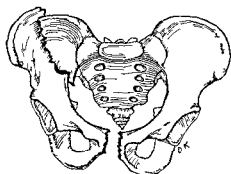


FIG. 56

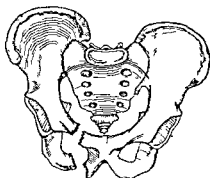


FIG. 57

Combined injuries of the pubic and iliac segments of the pelvic ring produced by antero-posterior compression. There may be severe displacement. Patients should be treated on the roads. (Bjork and of B. J. Jo. Surg. from the hospital.)

Mechanism of displacement in dislocation of the pelvis—Radiographic examination shows obvious separation of the two pubic bones but very slight displacement of the sacro iliac joint which may even be overlooked altogether. Only careful examination shows that the ilium overlaps the back of the sacrum more than on the normal side and that the iliac joint surface is slightly higher than the sacral joint surface. Similarly, on clinical examination it may almost be possible to put a fist between the displaced pubic bones and yet there is only a trace of undue prominence of the posterior superior iliac spine. This is because the displaced innominate bone is rotated round a longitudinal axis near the sacro iliac joint. The dislocated half of the pelvis is swung outwards and only secondarily in the more severe cases is it displaced upwards into the loin. This outward rotation is clearly shown in radiographs by the outwardly rotated position of the femur by the unusual prominence of the ischial spine and by the disappearance of the obturator foramen (Fig. 592). It is a displacement which is maintained by the weight of the limb.

Many types of treatment have been devised to correct the displacement. Traction has been applied to one or both limbs, skeletal traction has been used, well leg traction has been employed,¹ pelvic slings and girdles have been devised,^{2,4} operative wiring has been performed⁵ and a formidable operation has been suggested to expose both obturator foramina and force the two halves of the pelvis together by tightening a "grab hook" fixed on to the bones. In all these procedures the patient has been treated lying on his back.

The key to successful reduction is the position of lateral recumbency. The dislocated pelvis is like a partly opened bivalve shell when laid on the hinge at the back, gravity keeps the two halves apart, but when laid on one side the two halves close. Similarly, with the dislocated pelvis, if the patient lies on one side the two halves of the pelvis fall together.

TREATMENT OF DISRUPTION OF THE PELVIS

Watson-Jones postural reduction⁶—Slight separations of the symphysis pubis with subluxation of the sacro iliac joint may be reduced without anaesthesia if there is gross displacement spinal or general anaesthesia should be used. A plaster table or any form of pelvic rest is used with the perineal post removed. The patient is placed on his uninjured side with the ilium and trochanter lying on the pelvic rest and the two lower limbs held one above the other by an assistant. In many cases the dislocation is already reduced by the time the patient is in this position. If the pubic bones are not perfectly approximated and the posterior superior spine of the ilium is still unduly prominent, pressure is applied over the crest of the dislocated ilium pushing and rotating it downwards and forwards towards the normal half of the pelvis (Figs 578-580). Accuracy of reduction should be confirmed by taking radiographs before the plaster is applied. If necessary the patient may be laid on the injured side so that lateral compression is increased by the addition of body weight.

The iliac crests are protected with adhesive felt and a double plaster spica is applied. The spica must be closely moulded to the pelvis and lumbar region. The pelvic rest is then cut out, padding is inserted and the gap in the plaster is repaired. A post reduction radiograph is taken through the plaster (Figs 582-583). Throughout the period of recumbency the patient is encouraged to lie on one side. After four or five weeks the plaster may become loose, and a new spica is then applied, again in lateral recumbency. Immobilisation is continued for three months. Throughout this time regular exercises are practised to prevent stiffening of the knee joints and to maintain the tone of the quadriceps muscles.

Treatment of fracture-dislocation of pelvis—The same routine may be adopted when instead of a dislocation of both joints there is a dislocation of one and a fracture near the other. In these cases the fragments do not always lock in the reduced position with the security and accuracy which is observed

¹ Broomhead "Sacro-iliac Dislocation Reduced by Hoke's Traction" *Proc Roy Soc Med* 1933 xxiv, 576

² Böller "The Treatment of Fractures" 4th ed Bristol 1935 159

³ Conway "Fractures of the Pelvis" *Amer Jour Surg* 1935 xxx 69

⁴ Buegel "Luxations of the Os Innominateum" *Jour de Chir* 1917 xlix 536

⁵ Sommer "Traumatic Disruption of Symphysis" *Bruns Zeit z Klin Chir* 1937 cix, 60

⁶ Watson Jones "Dislocations and Fracture dislocations of the Pelvis" *Brit Jour Surg*, 1935 xxv, 773

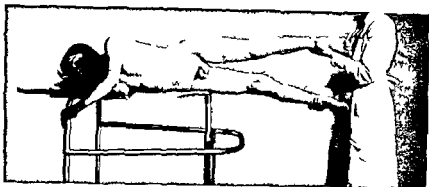


FIG 578

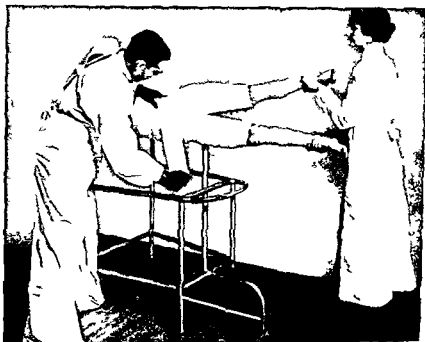


FIG 579

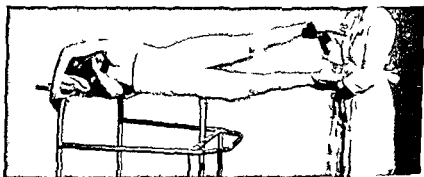


FIG 580

Reduction and immobilisation of dislocation and fracture dislocation of the pelvis by lateral recumbency. The patient lies on the uninjured side. The dislocated ilium is rotated forwards and downwards and a double plaster spica is applied. (*Brit Jour Surg* from the author's article 1938 xxv - 3)

in combined dislocations. The fragments are more mobile and over reduction is possible so that the pubic bones override.

Continuous traction after reduction by lateral recumbency—In addition to rotatory displacement there is sometimes upward displacement of the innominate bone into the loin. While the patient lies in lateral recumbency this is reduced by thrusting downwards on the crest of the ilium. A plaster spica is then applied and continuous traction may also be used. The limb on the dislocated side is supported in a Thomas splint incorporated in the plaster after cutting off the ring. Either skin traction or skeletal traction from the head of the tibia is used, the foot of the bed being raised on blocks.

Treatment of dislocation of pelvis with rupture of bladder¹—If disruption of the pelvis is complicated by extra peritoneal or intra peritoneal rupture of the bladder, the co-operation of a urological surgeon must be sought. Immediate operation is necessary and the bladder and prevesical space are drained. Leakage of urine is controlled by a tied-in suprapubic catheter and suction apparatus or by a Hamilton Irving box with rubber tubes leading to a receptacle. For the first few days a pelvic sling supported by weights over an overhead beam may be used to prevent lateral spreading of the pelvis, but after that time the dislocation should be reduced by lateral recumbency and immobilised in plaster (Figs 584-585). The injury to the bladder, the prevesical tissues and the pubis is one plane of rupture through the tissues of the midline and the two halves of the bladder wall remain attached to the corresponding halves of the pelvis. Infection in the prevesical tissues may increase the fixation of the bladder wall to the bone. If the pubic bones remain in their dislocated position all the soft tissues of the front of the pelvic cavity are held apart (Fig 581). Moreover every time the patient is turned the pelvic bones are pushed together and then forced apart and sutures in the bladder wall may tear through. It is clear therefore that reduction of the pelvic dislocation assists in approximating the rupture of soft tissues and that immobilisation of the pelvis promotes repair of the visceral injury. Leakage from the cystotomy wound is controlled during application of the plaster by a Malecot or de Pezzar catheter. A generous window is cut in the midline to expose the abdominal wound and to allow a Hamilton Irving box to be fixed in position.

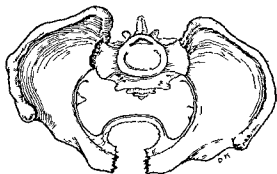


FIG 581

Dislocation of the pelvis with rupture of the bladder. The two halves of the bladder are held apart by the two halves of the pelvis and there is no satisfactory approximation until the dislocation is reduced.

Treatment of dislocation of pelvis with rupture of urethra—Reduction and immobilisation of the pelvic bone injury may also assist the urological surgeon with his treatment of an injury to the urethra. In some cases it has proved quite impossible to pass a catheter through the urethra into the bladder while the pelvis was dislocated, whereas as soon as the dislocation was



FIG 582

Dislocation of the left side of the pelvis. The rotation of the ilium is shown in the displacement of the left symphysis away from the midline, the prominence of the ischial spine, the disappearance of the obturator foramen and the abnormal overlap of iliac and sacral shadows. The arrow points to the lower margin of the sacral articular surface.



FIG 583

Same case as Fig. 449 twelve months after reduction by simple lateral recumbency and double plaster spica. The separation of the symphysis is corrected and the sacro-iliac displacement is reduced.



FIG 584

Dislocation of the right side of the pelvis with rupture of the bladder. The arrows point to the lower margin of the sacral joint surface on each side



FIG 585

Same case as Fig 584. Radiograph through plaster taken in operating theatre with portable unit. A de Lezgar catheter is preventing leakage from the bladder. Although it is three weeks since injury the dislocation is reduced. Plaster was retained for three months and despite continued bladder drainage prevesical infection and osteomyelitis of the pubis there was no serious nursing difficulty.

reduced by laying the patient on one side, a catheter was passed without difficulty. The replacement of the bones has replaced the soft tissues, and kinking and distortion of the urethra has been relieved.

INJURIES OF THE SACRUM AND COCCYX

Fracture of the sacrum—Extensive crushing injuries of the pelvis are sometimes accompanied by fractures of the sacrum. Isolated fractures are rare, and the injury is usually a crack fracture without displacement which recovers rapidly and completely. Occasionally the lower half of the sacrum is displaced forwards into the pelvic cavity, and there may be injury to the distal sacral nerves with saddle anæsthesia of the gluteal regions and incontinence. An attempt should be made to replace the bone by manipulation with one finger in the rectum.

Fracture of the coccyx—A fall in the sitting position may cause a contusion, a fracture, or a dislocation of the coccyx. If the distal fragment is completely separated it is pulled forwards by the ano coccygeal and levator ani muscles. The injury always causes considerable pain which may persist for several months. There is difficulty in sitting and the only comfortable positions are standing and lying. No special treatment is necessary except to minimise pain by protecting the bone from further injury. It is wise to warn the patient at the beginning that the injury is like any other fracture in that its repair may occupy weeks and sometimes months, and that even if some pain should persist so long, it is still of no serious significance and it is no more than has been expected. If this is done the patient usually accepts the position. Functional disorders do not arise, and recovery is quite complete within three or four months. On the other hand, the patient who is told that the injury is trivial and whose complaint of persistent pain is ignored, becomes anxious, fears complications and may develop superimposed functional disorders.

Operative treatment—The slowness of repair is accounted for by the pull of many muscles inserted into the coccyx, they strain the line of injury every time the patient moves, sits or defæcates. For the same reason pain sometimes persists indefinitely. These symptoms are relieved when the coccyx is excised. If the operation is done with care, the whole coccyx removed and the stump of the sacrum rounded the results are entirely satisfactory.^{1,2} The functional and psychological aspect of coccydynia has been exaggerated. The victims of this disorder exhibit few or none of the usual manifestations of functional disturbances and if their complaint of pain is accepted and treated as it should be their recovery is usually quite complete.

¹ Lewin The Coccyx—its Derangements and Treatment *Surg Gyn Obst* 1920 xlv 705
² Johnson "Derangements of the Coccyx" *Nebraska State Med Jour* 1936 xxi 451

CHAPTER XX

INJURIES OF THE CHEST

Children and adolescents seldom sustain fractures of the ribs, but the incidence of the injury increases with advancing years as the chest wall becomes more rigid. It must be recognised that the rarity of rib fracture in children is due to the elasticity and flexibility of the chest wall and that this is a factor which tends to increase the risk of visceral injury. The degree of internal injury cannot be judged by the injury to the chest wall. Similarly in adults it is fallacious to assume that a trivial rib fracture cannot be associated with more than a trivial lung injury.

FRACTURES OF THE RIBS

Mechanism of injury—A direct blow due to a fall against the edge of a table or the side of a bath may produce a crack fracture of the exposed parts of the ribs in front of the midaxillary line. Crushing injuries seldom fracture the ribs at the site of violence. More commonly the ribs give way at the point of maximum convexity in the region of the costal angle. The first and second ribs are often protected by the clavicle and the eleventh and twelfth ribs usually escape. In more severe cases however the whole thoracic framework is crushed several ribs may show double fractures and there may also be fractures of the clavicle and of the lumbar transverse processes. Rib fractures have been reported from simple muscular strain^{1 3}. Violent sneezing and coughing may be responsible especially in severe tracheitis and in whooping cough. The injury sometimes occurs from the muscular effort of lifting heavy loads from the strains of childbirth and even from the exertions of golf.

Symptoms—There is pain at the site of injury, tenderness on pressure and pain on antero posterior compression of the chest. Respiratory movements are short and shallow. Patients are usually more comfortable when sitting than when lying. The intercostal tissues provide a considerable degree of natural immobilisation and union is rapid. Callus forms within ten or twelve days. There may not be radiographic evidence of consolidation of the fracture until several months after injury but functional recovery is usually complete within a month or six weeks.

Treatment—During the first week or two aggravation of pain by respiratory movement may be relieved by strapping. Long strips of strapping

¹ Palfrey Fractures of Ribs (ten cases due to coughing) *Boston Med and Surg Jour* 1904 cxcl 493

² Jones Rib Fracture produced by Sneezing *Glasgow Med Jour* 1907 lxvii 206

³ Kleiner Fracture of Rib by Muscle Action (review of fifty six reported cases) *London Med and Surg Jour* 1904 cxv 1034

extending across the midline both in front and behind are applied while the chest is held in the position of expiration. The upper ribs may be supported by strapping from the region of the nipple over the clavicle to the lower angle of the scapula. If the pain is still unrelieved the chest should be completely encircled so that respiratory movement is almost confined to the diaphragmatic and abdominal muscles.

Novocaine injection for fractures of the ribs—During the last few years success has been claimed for the treatment of fractured ribs by novocaine injection. Pain is relieved and strapping of the chest is unnecessary. This freedom from symptoms may be attributable to the relief of muscle spasm or it is explained by the theory of Leriche (p. 362). The point of maximum tenderness is localised and the overlying skin is anaesthetised. A wider bored needle is then slid along the surface of the rib until the fracture is felt. From 5 to 10 c.c. of 1 per cent novocaine is injected into the fracture hæmatoma and the surrounding muscles. The same procedure is repeated at other fracture levels.

'Stove-in' chest—A severe crushing blow may fracture several adjacent ribs anteriorly and posteriorly so that a segment of the chest wall is completely separated. Paradoxical respiration occurs: as the chest expands in inspiration the floating part retracts; with expiration it expands. If a large part of the chest wall is involved air cannot enter the lung or be expelled. Light sandbags or a tight dressing should be applied. It is sometimes necessary to use an artificial respirator.

SLIPPING RIB

Recurring luxation of a costo chondral joint causes pain which is often attributed to neuritis, pleurisy, angina, coronary thrombosis, cholecystitis or kidney disease and sometimes to neurasthenia or malingering.¹⁴ There is often a history of injury followed by persistent pain which is severe and frequently radiating. The pain is aggravated by abducting the arm, playing golf or lifting weights. There is local swelling and tenderness over the costo chondral junction; the rib or cartilage may be unduly prominent and appear to be subluxated; and recurrent subluxation may be proved by the clicking sensation which accompanies deep respiration, movement of the trunk or movement of the arm. The symptoms are sometimes relieved by novocaine injection or by strapping the chest.⁵ Manipulative treatment has been credited with dramatic results.⁶ If symptoms persist it is wise to cut down on the joint and excise the costal cartilage or the end of the rib.

FRACTURES OF THE STERNUM

Fractures of the sternum accompany severe fracture dislocations of the thoracic spine but in these cases the injury is usually fatal. Isolated fracture of the sternum is rare but it may occur in motor accidents.

R. Du Bois, Coley, R. B. Mahon, C. J. Marshall, F. J. Poynton, E. N. Russell, H. K. V. S. Shaw, B. P. 1928
 Riba, B. H. Mel. J. 1919, 1, 43, 516, 60 and 664
 L. E. C. H. V. Practitioner 1919, 3, 314
 D. B. Vard. Slipping Ribs, Jo. Am. Med. Ass. 1931, 96, 111
 E. Graham, J. Singer and H. C. Balbon, Surg. Cal. D. Soc. of the Cl. t. Lea & t. blz. r. 11, 11, 17, 193
 H. C. Balbon and J. Spector, Slipping Ribs, Canal Med. Ass. Jo. r. October 193, 35
 W. A. 1, 1, D. pl. ced. R. 19, 1931, 11

particularly from the impact of the steering column against the front of the driver's chest.¹ The junction of the mobile body of the sternum with the relatively fixed manubrium sterni is the usual site of injury and there is backward displacement of the body sometimes with overriding of the fragments. Lateral and oblique radiographs taken in the position of inspiration show the injury (Fig 586). The spine should be hyperextended by pillows beneath the upper dorsal spine or if necessary by a hyperextended frame or plaster jacket. Union is firm in six weeks. Rupture of the internal mammary artery with hæmothorax has been described as a complication.



FIG 586

Fracture of the sternum at the junction of the manubrium with the body which is displaced backwards

FRACTURES OF THE HYOID BONE AND THYROID CARTILAGE

Compression fractures of the thyroid cartilage and hyoid bone are fortunately rare for the mortality may be as high as 75 per cent.³⁴ The thyroid cartilage is crushed against the vertebral column by a blow or it is compressed laterally in hanging and strangling. The injury usually occurs in adult men. It is very rare in women whose laryngeal cartilages seldom ossify. The greatest danger is from œdema of the larynx. A tracheotomy set must be available for instant use during the first ten days. Excessive salivation causing painful swallowing should be controlled with atropine. Talking is forbidden and nasal feeding may be used.

VISCERAL COMPLICATIONS OF CHEST INJURY

Crushing injuries of the chest cause (1) traumatic asphyxia (2) pneumothorax due to laceration of the lung and bronchi or a punctured wound of the chest wall (3) hæmothorax due to laceration of the lung or rupture of the intrathoracic systemic vessels (4) intrapericardial hæmorrhage (5) rupture of the diaphragm and diaphragmatic hernia.

Traumatic asphyxia—Compression of the chest due to a crushing injury may force blood back from the heart and intrathoracic veins into the innominate internal jugular and external jugular veins. These vessels are not protected by functioning valves. The smaller veins and venules are therefore distended and blood may also be extravasated into the tissues of the head, neck and upper chest thus causing temporary loss of consciousness.

¹W. Stuck "Fractures of the Sternum" *Amer Jour Surg* 1933 xlii "66 and 1937 xxxviii 560
²Holterman "Fracture and Dislocation of the Sternum" *Ann of Surg* 1924 lxxviii 2.
³Stuck "Fractures of the Thyroid Cartilage" *Amer Jour Surg* 1917 xxxv 156.
⁴Heenberg "Fracture of Hyoid Bone" *Ann of Surg* 1931 xlii 547.



FIG 587

Traumatic asphyxia

A crushing chest injury drives blood from the chest into the veins of the neck and head thus causing sudden distension of venules and extravasation of blood into the conjunctivæ and the skin except where it is supported by the pressure of braces collar and collar stud (Taken by Squad on Lead II Coleman)

ness bleeding from the nose and ears subconjunctival hæmorrhage pigmentation of the skin and often pulmonary congestion with radiographic appearances resembling milary tuberculosis. The skin pigmentation is brick red in colour. Owing to its distribution in the face and neck gradually fading out over the upper chest it was described by Ollivier as the *masque ecchymotique*. The blood is driven into the tissues by relatively low intravenous pressure and extravasation into the skin is therefore prevented by any external support such as the pressure of braces collar or even a collar stud. On the other hand in the loose subconjunctival tissues where resistance is least extravasation is most pronounced.

Traumatic asphyxia may follow crushing of the chest due to panic in a crowd. Six cases were admitted to the Bellevue Hospital New York after



Fig 588

Traumatic asphyxia. There is no blanching under the pressure of a glass spatula thus suggesting that the discoloration is not due to simple venous congestion (but see footnote¹)

the cry of fire had been falsely raised in a neighbouring cinema. The patient illustrated in Figs 587-588 was an airman who was pinned under the framework of an overturned 30 cwt lorry the weight being across the upper abdomen and lower chest.² He was released within two minutes. He recalls a sensation of pressure across the abdomen spreading up as if the blood was being forced to his head. He then lost consciousness. Ten minutes later when being lifted into the ambulance he regained consciousness but complained of blurred vision as if looking through a mist. His friend noticed that he was breathing heavily and slowly and that his chest was red and his face turning purple. When examined shortly afterwards there was bleeding from both ears with hæmorrhage on the

¹ Is the pigmentation due to extravasation of blood in the subconjunctival tissues and the nasal mucosa where external support is minimal. Moreover, the absence of blanching under the pressure of a glass spatula would suggest that pigmentation of the skin is also due to extravasation (Fig 584). But the color is very different from that of a bruise or the ecchymosis of a fractured bone. Moreover it fades and disappears within two or three weeks without going through the color changes of extravasated blood. Act ally the glass spatula test is not conclusive of extravasation. The blood might be trapped in acutely dilated venules so that it cannot be dispersed by external pressure although it is not in normal circulation it can gain a feeble supply of oxygen thus explaining the brick red rather than deep blue color and the absence of yellowing and green pigmentation due to the breakdown of hemoglobin by the action of leukocytes. This is confirmed by microscopic section of the skin where a striking venous congestion but no more than normal or all local areas of extravasation. (L. H. Gray, War Injuries of the Chest, *Proc Mayo Clinic* No. 194, xvii, 36, 366.)

² H. Colman, Traumatic Asphyxia, *Proc Roy Soc Med* 194, xiv, 1.

drums and subconjunctival hæmorrhage with exophthalmos and œdema of the lids. Blurring of vision persisted for three hours. Two ribs had been fractured and pulmonary œdema and congestion developed. The skin pigmentation gradually faded but without going through the usual colour changes of a bruise. It was normal within three weeks. The conjunctivæ still showed staining after two months. Recovery was complete with no loss of hearing or vision.

Open pneumothorax¹⁴—A large wound of the chest wall allows communication between the pleural space and the atmosphere, destroys the pleural negative pressure and causes collapse of the lung and anoxæmia. But this is not the most serious effect of an open pneumothorax. The greatest danger arises from pendulum action of the mediastinum which is sucked to and fro with every respiratory effort. The right side of the heart does not fill normally and a patient with a large sucking wound cannot survive long. As a first aid measure the lung should be grasped with any suitable instrument and retracted into the wound of the thoracic wall so that the pendulum swing of the mediastinum is controlled. Subsequent treatment is discussed on p. 270.

Tension pneumothorax—If a wound of the chest wall or of the lung is valvular so that air enters the pleural cavity on inspiration but cannot be expelled during expiration a tension pneumothorax develops. There is increasing intrapleural pressure, displacement of the heart and mediastinal structures to the opposite side and pressure on the right side of the heart and great veins with increasing dyspnoea and cyanosis. These are urgent indications for aspiration of air from the pleural cavity. A short wide bore needle is inserted through the second intercostal space 2 in. from the margin of the sternum and connected to a tube which passes under sterile water in a bottle below the level of the bed, thus allowing escape of air but preventing its re-entry. The needle and tube are left in position until air no longer bubbles through the fluid, showing that the communication between pleura and lung is closed.

Subcutaneous and mediastinal emphysema—A tension pneumothorax may cause subcutaneous emphysema due to the mechanical effect of muscular activity forcing air through a tear in the parietal pleura. The emphysema may extend as far as the scrotum and upper thighs. Mediastinal emphysema is due to a tension pneumothorax with rupture of the mediastinal pleura or it is due to rupture of a primary bronchus or the trachea. Air is forced into the mediastinum and emphysema spreads to the upper chest, neck and face.

Hæmorthorax—Hæmorthorax may be due to laceration of the lung or rupture of the intercostal or mammary vessels. In the pulmonary arteries the blood pressure is no more than a quarter the pressure of the systemic circulation so that spontaneous arrest of hæmorrhage is much more likely to occur after laceration of the lung than after laceration of systemic arteries. The level of blood in the pleural cavity must be closely observed. If it does not rise above the nipple line there is no serious embarrassment but if despite conservative treatment bleeding persists it is probable that

Tudor Edwards. Treatment of Injuries of the Chest. *Brit. Med. Jour.* November 1933. 1936.

* Sauerbruch and O. Shalghessay. "Thoracic Surgery." London, 1934.

* Butler. Injuries of Chest and Abdomen. *Surg. Gyn. Obst.* 1933. ix. i. 418.

Boland. Traumatic Surgery of Lungs and Pleura (review of 1197 cases of wound). *Ann. of Surg.* 1926. clv. 2.

the internal mammary or one of the intercostal vessels is torn and operative intervention is necessary. In non progressive hæmothorax the blood should be aspirated after three or four days in order to prevent infection. It should be removed slowly, and be replaced by an equal quantity of air in order to prevent sudden re inflation of the lung with recurrent hæmorrhage.

Hæmoptysis after fracture of the ribs and crushing injury of the chest is usually due to rupture of small pulmonary vessels by the contusion. It does not necessarily indicate penetration of the lung by rib fragments.

Empyema—When injury of the lung is associated with pleural effusion and hæmothorax there is usually a febrile reaction for several days, and a temperature of 100° or 101° F does not necessarily mean that there is an empyema. The effusion should be aspirated on alternate days. Open drainage is not indicated unless the aspirated fluid becomes frankly purulent.

Wounds of the heart¹ causing effusion of blood into the pericardium, rising intrapericardial pressure and "cardiac tamponage" may be recognised by the three classical signs (1) muffling of heart sounds, (2) falling blood pressure (3) rising venous pressure. If the intrapericardial pressure exceeds 200 mm there is cardiac embarrassment and increasing pressure causes death. The pericardium should be exposed to the left of the sternum between the third and fourth costal cartilages, the pleura is retracted, the pericardium opened and the wound sutured.

¹ R. A. Griswold and C. H. Maguire
1942 lxxiv 406

Penetrating wounds of the Heart and Pericardium *Surg Gyn Obst*,

CHAPTER XXI

FACIO-MAXILLARY INJURIES

Recent progress in the treatment of facio maxillary injuries has been achieved by co operation and team work, and by application of the principles of orthopaedic surgery, the technique of plastic surgery and the mechanics of dental surgery¹⁷ Co operation between specialists is particularly important in time of war when simple fractures due to blows and falls are relatively infrequent, and the majority of facio maxillary injuries are the result of bomb or shell explosion collapse of buildings, collision of motor cycles and crash of aircraft

EMERGENCY TREATMENT OF FACIAL WOUNDS AND FRACTURES

The reconstructive treatment of deformity and disfigurement of the face is the responsibility of the skilled plastic surgeon and his team but every casualty surgeon must be prepared to undertake the emergency treatment of facial wounds and fractures

Excision of wounds—The whole face should be washed and grease or oil removed with ether and ether soap Hair may be cut short, but it should not be shaved from the eyebrows or hairline of the scalp where it serves as a guide for the accurate apposition of skin flaps Pigmentation of scars must be prevented by cleaning the injured tissues thoroughly with soap and water by scrubbing ingrained dirt with a tooth brush and when necessary by excising tattooed tissues with a scalpel or sharp curette The technique of operation is similar to the excision of wounds in other regions (Chapter X) except that the face and jaws are so freely supplied with blood, the resistance to infection so high and the risk of gas gangrene so negligible that the operation is much less radical Very light trimming of the skin margin is all that is needed, flaps which are almost completely detached are often viable, excision of soft tissues should be limited, bone fragments which have the slightest chance of survival are not removed Hemostasis is secured by Spencer Wells' forceps and torsion, the larger vessels being ligatured with fine catgut The wound is lightly powdered with sulphanilamide

Suture of skin—In view of the free blood supply and the high resistance to infection, suture of the skin is advisable when the operation is performed

¹⁷ R. H. Ivy and L. Curtis "Fractures of the Jaw" London 1934

¹⁸ W. Warwick James and B. W. Pickling "Injuries of the Jaw and Face" London 1943

¹⁹ Kelsey Fry and others "Dental Treatment of Maxillo-facial Injuries" London 1942

²⁰ Kurt H. Thoma "Traumatic Surgery of the Jaws" London and St. Louis 1941

²¹ "Manual of Standard Practice of Plastic and Maxillo-facial Surgery" Philadelphia and London 1942

²² "Report on Maxillo-facial Injuries" London 1935

²³ P. P. Cole "War Injuries of the Face and Jaws" War Wounds and Injuries Edited by L. E. Ketcher and

R. W. Raven London 1943

within twelve hours of injury. Care must be taken to secure exact apposition of skin flaps and to minimise the disfigurement of suture marks. Very fine material should be used such as ophthalmic silkworm gut, kaldernic or nylon sutures mounted on eyeless needles. The needle should be inserted close to the skin margin. The sutures must not be tied tightly. The wound is drained by means of a few strands of silkworm gut twisted together, inserted between stitches and removed after twenty-four hours. Alternate stitches are removed on the third day and the remainder on the fourth or fifth day. It may be safe to suture the skin even up to twenty-four hours after injury but if there is tension on the flaps or a probability of infection, it is better to leave the wound lightly packed open with gauze. Secondary suture may be performed within a few days and even when this is not possible and the wound heals by granulation the resulting scar is less conspicuous than that which follows infection of a sutured wound. Moreover, it can be excised and sutured at a later date whereas the disfiguring marks of badly placed sutures which have cut through the tissues are much more difficult to remove.

Full thickness loss of skin—In penetrating wounds of the face with destruction of bone and full thickness loss of the cheek the mucous membrane of the buccal surface should be sutured to the skin edges. In this way the fractured surface is covered, bone and soft tissues which would otherwise be exposed are protected from infection and secondary deformity is minimised. When infection is controlled the soft tissues are reconstructed by plastic repair and the defect in the bone is made good by grafting.

Extraction of teeth—The decision whether or not to extract teeth which lie in the line of a fracture of the jaw¹⁴ must be based on three considerations. (1) occlusion of teeth is a reliable test of accurate reduction of a fracture and even when it is necessary to extract teeth their occlusion should be tested first. (2) fixation of teeth is used for the attachment of wires and splints in the immobilisation of fractures and no tooth should be extracted unnecessarily when a single tooth remains on one fragment it must be retained as long as it is of value for immobilisation. (3) infection causes sequestration of teeth in the line of a fracture with persistent infection and delayed union the tooth must be removed sooner or later. In general, therefore it is wise to be conservative in the extraction of teeth in simple fractures but to remove all loose and infected teeth in compound comminuted fractures.

Immobilisation of injured tissues—Immobilisation not only promotes the union of fractures but also assists the healing of soft tissues, prevents the spread of infection and controls the development of deformity. While the surgeon is engaged in excising and suturing the wound and reducing the displacement of fragments the dental surgeon takes impressions of the teeth and subsequently prepares fits and adjusts the splints. If skilled dental assistance is not available three simple methods of immobilising the jaw may be used. (1) the patient's dentures may be put in position with the jaw strongly supported by means of a firm bandage. (2) the lower denture may be fixed to the mandible by circumferential wiring, (3) the

¹⁴ Kelsey Fry, Fractures of the Mandible, *Proc Roy Soc Med* 1928, xxii, 663

¹⁵ W. W. James and E. Kling, Injuries of the Jaw and Face, London, 1941

¹⁶ G. J. Larratt, Teeth in the Line of Fracture, *Br Dent Jour* 1945, lxxix, 11

¹⁷ Walker, Should Teeth in the Line of Fracture be removed? *Proc Roy Soc Med* 1942, xxxv, No. 10, 663

teeth in the upper and lower jaws may be ligatured to each other by interdental wiring. In bandaging the jaw it is important to secure vertical support and to avoid the backward pull of a four tailed bandage which is liable to cause receding of the jaw and mal union of fractures.

Preventing infection—Throughout the period of immobilisation special attention must be paid to the control of infection of the mouth. Rigid toilet of the teeth and antiseptic irrigation of the mouth is necessary (Fig 589). A fluid or soft diet is prescribed.

FRACTURES OF THE MANDIBLE

If the jaw is struck while the teeth are clenched the mandible usually escapes fracture because the force is transmitted directly to the skull and to the powerful muscles of the neck. but if the jaw is in the normal position of rest and the teeth are slightly apart similar violence is likely to fracture the bone. A blow on the point of the chin may cause bilateral fractures of the condyles. if it is sufficiently forcible it also splits the body of the mandible near the symphysis (Fig 593). Blows on the lateral aspect of the jaw tend to fracture the bone at the site of impact and near the canine region on the opposite side (Fig 594). Fractures of the angle of the jaw (Fig 591) the ascending ramus and the coronoid process occur less frequently.¹

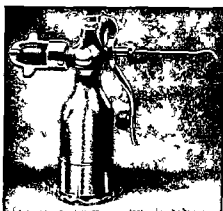


FIG 589

Simple type of dental spray with CO₂ sparklet used for mouth washes in fracture of the mandible

The fragments of a fractured jaw may be displaced by the initial violence and deformity is increased by muscle traction. Muscles attached to the body of the bone pull downwards whereas muscles attached to the ramus pull upwards. In a bilateral fracture of the body of the mandible the chin fragment is therefore depressed. in a unilateral fracture near the posterior part of the body the posterior fragment is rotated upwards.

Clinical features—There is local swelling, bruising and deformity, often with dribbling of blood stained saliva from the open mouth, fetor of breath and difficulty in articulation and swallowing. Anaesthesia of half the lower lip points to a fracture crossing the inferior alveolar canal behind the mental foramen. Movement of the jaw is restricted, there may be deviation to one side, irregularity of the teeth and inaccurate occlusion with the teeth of the upper jaw. Bruising of the mucosa of the cheek, gums and floor of the mouth indicates a fracture of the body. In the edentulous mandible displaced fractures of the body are usually compound and extend into the mouth. Bruising of the tonsillar pillars of the lower part of the soft palate and of the lateral pharyngeal wall point to a severe fracture of the vertical portion of the bone. The diagnosis is confirmed by radiographic examination which should include three standard views—postero anterior skull, right lateral mandible and left lateral mandible. Additional projections which may be

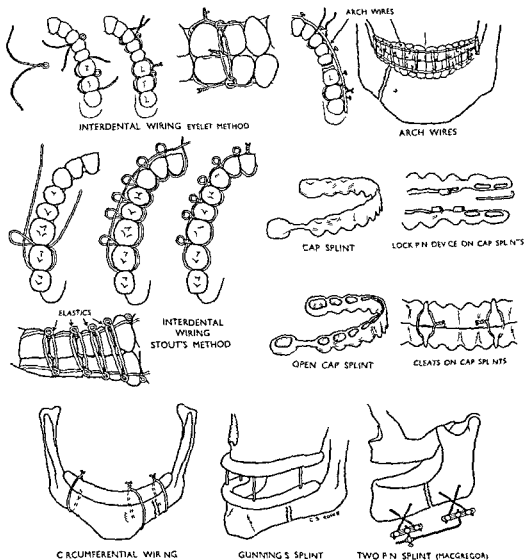


FIG 590

When there are teeth in both fragments a fracture may be immobilised by interdental or arch wiring or still better by cap splints. When the jaw is edentulous Gunning's splint, circumferential wiring or the two pin splint is used.

of value include lateral views of the temporo mandibular joint with the mouth open and the mouth closed dental and occlusal views and the 30° oblique view or Stenver's projection

Methods of immobilisation—The mandible may be immobilised (1) when teeth are present by interdental wiring or cap splints (2) when the jaws are edentulous by the patient's dentures Gunnings splint or circumferential wiring (3) by the two pin skeletal transfixion splint (Fig 590)

Interdental wiring—Non-corrosive steel ligature wire should be used A



Fig 91

Fracture of the mandible immobilized by interdental wiring

1 m length is doubled round the closed points of haemostat forceps and twisted to form a small central eyelet Both ends of the wire are passed through the dental interspace selected leaving the eyelet on the labial side The two ends are then brought back through the next interspaces on each side and twisted tightly together below the eyelet in the upper jaw and above the eyelet in the lower jaw

Several pairs of ligatures are fixed in the upper and lower

jaws directly opposite to each other or in oblique relationship so that when they are wired together in oblique pull will be secured When all ligatures are in position the fracture is reduced by manipulation each opposing pair of eyelets is then secured by passing wire through the loops and twisting it tightly (Fig 91) Tightening and adjustment of these connecting ligatures may be required from time to time this being done without moving the eyelets themselves The wires should be protected from breaking by the additional support of a firm jaw bandage

Arch-wire method—The technique of interdental wiring has been facilitated and improved by the introduction of arch wires These are curved metal rods which can be fixed accurately to the teeth of the upper and lower jaws by means of dental wiring They are placed on the buccal side of the teeth near the necks and are fitted with studs by which the two arch wires can be secured to each other with simple wires or elastic bands Stout's method is illustrated in Fig 590



Fig 92

Fracture of the mandible immobilized by cap splints and block

Cap splinting—Interdental wiring and arch wiring are useful emergency measures and when there is a good complement of teeth and little tendency to displacement they may suffice for the whole treatment. But these methods fail when there is marked tendency to displacement. The fixation is not sufficiently stable, constant readjustment and tightening is necessary and the wires are liable to break. More reliable fixation can be secured with the aid of a skilled dental surgeon by cap splints made of cast metal or



FIG. 593

B lateral fractures of the condyles and fracture of the symphysis immobilised by cap splint

acrylic resin (vulcanite substitute). The splints are specially constructed to plaster casts of the teeth and are cemented to them. They give positive and stable immobilisation. With the incorporation of locking devices flanges and hooks many of the problems which are difficult of solution with wiring methods are easily overcome. Splints applied to the upper and lower jaw may be fixed to each other by a simple lock pin (Fig 592). When properly applied the splints do not harm the teeth or gums. They are applicable to all except edentulous cases.

Dentures Gunning's splint and circumferential wiring—In edentulous cases it may suffice to fit the patient's dentures and add the fixation of a firm jaw bandage. Alternatively the surgeon may use Gunning's splint which is an upper and lower denture joined together by vulcanite blocks

or metal rods to form one piece. But these methods are not always successful in preventing redisplacement, and if the fracture unites with deformity it may be impossible for the patient to wear dentures with comfort. The method of circumferential wiring of the mandible and denture was therefore introduced. On the posterior fragment $\frac{3}{4}$ in from the fracture line a point is marked on the buccal sulcus at the reflection of gum to cheek. A Reverdin needle or trocar and cannula is passed close to the bone, emerging through the skin at the lower border of the mandible. Stainless steel wire, 0.5 mm



FIG 594

Fracture of an edentulous mandible immobilised by Gunning's splint with circumferential wiring

diameter is guided through the cannula and pulled into the mouth. The cannula is withdrawn. At a corresponding point of the reflection of mucous membrane from the gum to the floor of the mouth the instrument is passed against the lingual surface of the bone to emerge through the same skin perforation. The other end of the wire is brought into the mouth. The wire is then sawed through the tissues until it lies in close contact with the mandible. In a similar manner two other wires are passed one $\frac{3}{4}$ in in front of the fracture line and one on the opposite side of the jaw. The patient's denture or a specially made vulcanite or acrylic plate is placed in position, the fracture is reduced and the three wires are twisted tightly over the plate (Fig 594).

External pin fixation by two-pin splint—The two pin splint is an application of the technique devised by Roger Anderson and Haynes for fractures

of the shafts of long bones (pp 162 and 186) It consists of a pair of two pin units one unit fixed in each fragment and secured to each other by means of a connecting rod¹⁶ The upper and lower borders of the mandible and the line of fracture are marked on the skin with Bonney's blue ink. An assistant holds the posterior fragment firmly near its lower border 1 in from the fracture line a pin is drilled obliquely into the bone at an angle of 60° a second pin is then driven in crossing the first at an angle of 30° to 40° Another pair of pins is similarly drilled obliquely into the other fragment of the fractured bone Universal clamping joints are attached firmly to the pins and the connecting rod is loosely attached The fracture is reduced the clamps on the connecting rod are securely tightened and the fragments are thereby immobilised in the reduced position (Fig 595)

Another skeletal transfixion method used in fractures of the long bones which has been applied to mandibular fractures is direct transfixion of the two fragments by Kirschner wires or heavier stainless steel wires drilled through puncture stab wounds and left in position with the wires projecting through the skin for a period of three to five weeks^{7,8}



FIG 595

Double fracture of the mandible immobilised by two pin splints one on each side of the jaw

Complications—The complications of fracture of the mandible include sepsis mal union non union and trismus *Sepsis* is controlled by drainage of the bone removal of sequestered bone and dead teeth and prolonged immobilisation *Mal union* due to failure of early treatment may cause mal occlusion and necessitate extraction of teeth and the fitting of dentures More serious deformity including mandibular protraction and retraction is treated by osteotomy followed by immobilisation in the corrected position⁹ *Non union* is usually due to extensive loss of bone with infection and imperfect immobilisation When quiescence of infection has been secured and a sufficient interval has elapsed a broad flat graft is cut from the crest of the ilium and laid across the gap in contact with at least 1 in of rawed

¹ H D Glicks Reinforcement and Control of Maxillo-facial Fractures *Brit Dent Jour* 1941 lxxi 11
² M A Ruiton and J A Walker Mandibular Fractures treated by Pin Fixation *Amer Jour Orthod and Oral Surgery* 1942 xxxvi 307
³ R Mowle J D Burton A B MacGregor J Barron External Pin Fixation for Fractures of the Mandible *Lancet* Oct 1941 391
⁴ R Mowlem Skeletal Fixation in Fractures of the Jaws *Brit Dent Jour* 1941 lxxi 10
⁵ P Mowlem Extra-oral Splinting of the Fractured Mandible *Lancet* Oct 1941 39
⁶ L Lohd *Extra-oral Splinting of the Fractured Mandible* *Lancet* Oct 1941 39
⁷ H Meade *Irish Jour Med Sc* 1915 718
⁸ J B Brown and E Melowell Wire Fixation Fractures of Jaw *Surg Gyn Obst* 1940 lxxiv 0
⁹ Hensel Correction of Mandibular Protraction and Retraction *Surgery St Louis* 1933 ii 9

bone on each fragment^{1 2} Fixation is secured by cap splints or one of the methods outlined above. Internal fixation by wires or screws should not be used because there is danger of recurrent infection. For the same reason Nowlem advocates that the graft should not include cortical bone. In the presence of infection cortical bone undergoes sequestration and causes persistent delay in union of the fracture. This is avoided by using only cancellous bone chips. If reliable fixation is secured by splints the fracture unites even if slight infection recurs. It is therefore unnecessary to wait many months before performing the grafting operation. It may be undertaken within a few weeks of healing of the wound. *Trismus* may follow fracture dislocation of the condyles or compound comminuted fractures of the ramus. It is sometimes necessary to use cap splints on the upper and lower teeth fixed with a screwing device by which the jaw can be opened gradually.

DISLOCATION OF THE MANDIBLE

When the mouth is widely opened the head of the mandible on each side slides forwards into an unstable position on the eminentia articularis. A relatively light blow on the chin then causes bilateral dislocation. The displacement may arise spontaneously by the pull of the pterygoid muscle during yawning and vomiting or while taking an unusually wide bite. Unilateral dislocation occasionally follows the unskilful use of a mouth gag.

Clinical features—The mouth is held rigidly open with the jaw projecting forwards in bilateral dislocation and towards the uninjured side in unilateral dislocation. Swallowing and articulation are difficult and saliva dribbles from the mouth. The head of the mandible may be felt in front of its usual position with a hollow behind it at the level of the tragus.

Treatment—The operator stands in front of the patient. With his protected thumbs passed into the mouth he presses the angles of the jaw downwards and at the same time raises the chin with his fingers outside the mouth. As a rule the displacement is corrected easily without anaesthesia. If the dislocation is of longer standing and there is unusual difficulty, pieces of cork may be placed between the molar teeth on which the jaw can be levered when the chin is elevated. A bandage which elevates and supports the chin is kept in position for ten or fourteen days, a four tailed bandage which pulls the chin backwards should be avoided.

FRACTURE OF THE CONDYLE AND FRACTURE-DISLOCATION OF THE MANDIBLE³

In fracture dislocation of the temporo-mandibular joint the head of the mandible is dislocated from the glenoid fossa and it is separated from the condylar process by a fracture of the neck of the mandible. The injury is often bilateral and it may be associated with other fractures of the mandible.

Treatment—If the fracture dislocation occurs as an isolated injury, successful reduction may be possible by the manoeuvre described for simple dislocation. If this fails three methods of treatment are available: operative reduction, excision of the head of the mandible, conservative treatment.^{4 5}

¹ Billington and Round. Bone Grafting of the Mandible. *Brit Jour Surg* 1916 xlii 49.
² Harkins and Fletcher. On the Grafts for Fracture of the Mandible. *Jour Amer Med Ass* 1913 lxxii 11.
³ D. G. Walker. Fractures of Ramus and Coronoid Process of the Mandible. *Brit Dent Jour* 1914 lxxii 11.
⁴ Kappas. Fracture dislocation Jaw (adoption of operative replacement). *Zentralbl f Chir*, 1914 lxi 814.
⁵ Stenberg. Fracture dislocation of Jaw (adoption of excision of the head). *Acta Chir Scand* 1914 lxxi 39.

No operation on the temporo mandibular joint is easy because there is vigorous hæmorrhage and it may be difficult to avoid injury to filaments of the facial nerve. The dissection which is necessary to expose the displaced head is liable to cut its ligamentous and muscular attachments, and by

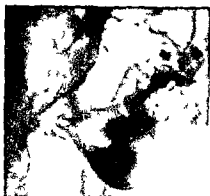


Fig 596

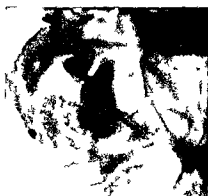


Fig 597

Bilateral fracture dislocation of temporo mandibular joints with a comminuted fracture of the body of the mandible—oblique views of the left and right temporo mandibular joints



Fig 598



Fig 599

Same case as shown in Figs 596 and 97 showing the free range of passive movement which may develop despite failure to reduce the fracture dislocation. This patient is now a physical training instructor in the army and has no disability at all.

depriving it of blood supply to cause avascular necrosis. Finally, unless the whole auricle is reflected forwards by an incision behind it a facial scar is inevitable. It is also to be recognised that the results of non-operative treatment are surprisingly good even when reduction fails (Figs 596-599). The best treatment therefore, is to attempt manipulative reduction and whether it succeeds or not, to support the jaw with the teeth in accurate

occlusion by means of bandage or cap splints and to permit active mobilisation within two or three weeks. Any tendency to lateral deviation of the jaw may be controlled even after mobilisation has begun by fitting a cap splint on the lower teeth with a flange against the upper teeth on the opposite side. If the resulting range of movement proves to be unsatisfactory or painful late excision of both mandibular heads can be considered.

Summary of the treatment of mandibular fractures—1 *Teeth in both fragments of lower jaw and in upper jaw*—Cap splinting or interdental wiring.

2 *Teeth in both fragments of jaw with loss of substance*—Through and through drainage and interdental wiring until cap splints are made.

3 *Teeth in both fragments of lower jaw but absent from upper jaw*—Cap splinting with upper denture for occlusion or two pin splint.

4 *Teeth in anterior but not in posterior fragment of lower jaw*—Cap splints or interdental wiring with a gutta percha mould pressing on the edentulous fragment or a two pin splint or wire traction on the posterior fragment to a rod connected with a plaster head cap.

5 *Edentulous mandible and edentulous maxilla*—Gunnings splint circumferential wiring or two pin splint.

6 *Fracture of ramus*—Immobilise for short period by cap splints with lock pin or interdental wiring or Gunnings splint.

7 *Fracture of condyle and fracture dislocation of jaw*—Permit early movement but prevent lateral deviation of the jaw by cap splint on lower teeth with flange against upper teeth on the opposite side.

CLASSIFICATION OF FRACTURES OF THE FACIAL BONES

The framework of the face is light and elastic. It consists of a series of bone plates which with the exception of the malar and palatal areas are thin and fragile. It is suspended from the skull by four vertical plates (the medial and lateral orbital walls) together with the zygomatic arches, pterygoid plates and lateral orbital margins. These bones are intimately connected as a complete facial skeleton and to consider them as separate bones is no more than an anatomical exercise. Injuries of the face should therefore be classified as fractures of regions and not as fractures of individual bones. Le Fort studied the effect of direct violence in experimental work on cadavers. He described three lines of weakness which form the basis of many classifications (Fig. 600). The following classification is modified from McIndoe¹—

1 Fractures of the naso maxillary unit

(i) Nasal fractures

(ii) Bilateral naso maxillary fractures

2 Fractures of the malar maxillary unit

(i) Impacted fracture of the malar

(ii) Comminuted fracture of the zygomatic arch

3 Fractures of the palatal unit

(i) Horizontal separation of the palate

(ii) Midline separation of the palate

(iii) Palatal alveolar fractures

FACIO-MAXILLARY INJURIES

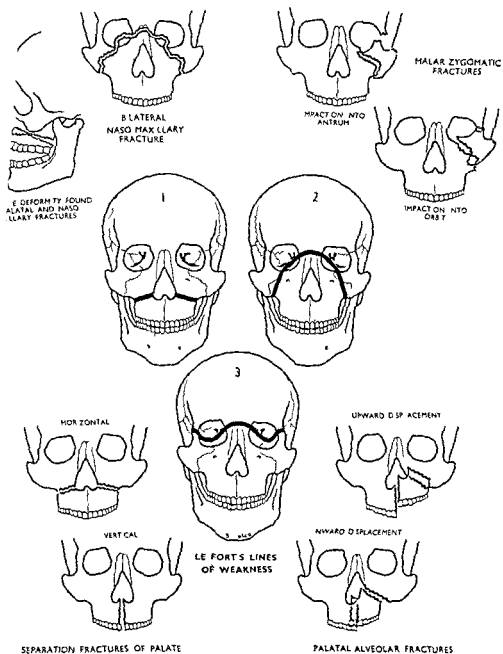
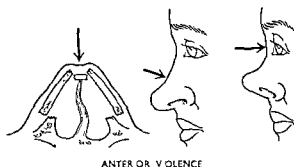


FIG 600

Classification of facio-maxillary fractures

FRACTURES OF THE NASO MAXILLARY UNIT

Nasal fractures—Deformity due to fracture of the nasal bones and cartilages is of four types according to the direction of violence (anterior or lateral¹) and the site of the blow (Fig 601) (1) When an anterior force strikes the nose near the glabella the nasal bones and adjacent nasal processes of the maxillæ are driven into the cavity of the nose the upper part of the septum and the ethmoid are damaged and the bridge of the nose is depressed. In profile the fronto nasal angle is lost and the tip of the nose is rotated upwards as seen from the front the nostrils are abnormally conspicuous.



ANTERIOR VIOLENCE



LATERAL VIOLENCE

FIG 601

Nasal fractures due to anterior violence cause depression of the nasal bridge lateral violence causes an S shaped deformity

(2) When the blow is sustained near the middle of the ridge at the junction of bone and cartilage the nasal bones are separated at the midline and driven back with their lateral margins overlapping the nasal process of the maxillæ so that the deformity includes not only flattening of the bridge of the nose but also broadening of the base (3) A blow at a still lower level causes fracture of the bony or cartilaginous part of the septum or dislocation of the septum from its bone ridge

on the vomer or from its attachment to the columellar cartilage. Dislocation from the columella gives rise to an obvious ridge within the nostril due to the displaced sharp septal margin (4) Lateral violence displaces the nasal ridge to the side towards which the force was travelling and gives rise to an S shaped deformity. The nasal bone which receives the blow is flattened its anterior margin being impacted beneath the opposite nasal bone which lies more vertically.

Treatment—Profuse hemorrhage may necessitate plugging of the nose as a first aid measure. Plugs must be removed as soon as possible and they should not be used at all if there is cerebrospinal rhinorrhœa. The deformity must be reduced under anesthesia. Simple manipulation with

the fingers may suffice to correct lateral displacement but if there is impaction of the fragments or depression of the bridge of the nose forceps are necessary. Ash's forceps are inserted with one blade on each side of the septum so that the deviation of the septum is corrected and the bridge of the nose elevated. Long bladed forceps (Walsham's forceps) protected with rubber tubing with one blade inside and one outside the nose are then used to correct the position of the nasal bones. When the deformity is completely corrected reduction is often stable without external support but it may be wise to use small plaster slabs 1 in. by 3 in. or to apply lateral pressure by gutta serena moulds fixed to a plaster head piece. If intranasal support is needed padded finger cots should be inserted in the nostrils for about twenty-four hours. Occasionally a special intranasal splint may be required (Fig. 602).

Bilateral naso-maxillary fractures—

These fractures follow the second line of weakness described by Le Fort (Fig. 600). The maxilla is displaced backwards between the malar bones thus causing malocclusion of the teeth. The upper teeth lie behind the lower and there may also be upward rotational displacement of the maxilla so that when the jaws are closed only the molar teeth meet—the open bite deformity. There is very extensive bruising and swelling of the face which in early days conceals the deformity. Diplopia is common.

Treatment—Nasal impaction is corrected by means of Ash's and Walsham's forceps. The maxillary block is then gripped with special forceps designed with spikes by which to secure a firm hold of the alveolar bone¹. The bone is rocked until impaction is released. Reduction of the fracture is maintained by a maxillary cap splint cemented to the teeth and fixed by means of a rod and universal joint (Walker Clouston joint) to a plaster head cap² (Figs. 603, 607). Mowlem has used cheek wires



FIG. 603

Watson Williams splint of light wire protected by rubber to prevent redispacement of vertical impact on fractures (as modified by Cerrero). *Canad. Med. Jour.* 1934 xxx.

¹ H. M. Indoe, D. D., and Treatment of Injuries Middle Third of Face. *Br. J. Dent. Surg.* 1941, lxxi, 7.
² R. Mowlem, Fixation Methods from the Standpoint of the Plastic Surgeon. *Br. J. Dent. Surg.* 1941, lxxi, 10.

attached to the mandibular and maxillary teeth and passing through the substance of each cheek to a plaster head cap¹ (Fig 605)

FRACTURES OF THE MALAR-MAXILLARY UNIT

Impacted fractures of the malar and comminuted fractures of the zygomatic arch² are due to lateral violence. They

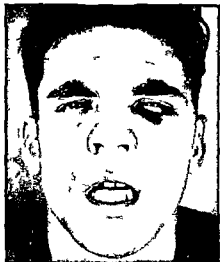


Fig 603

Depressed fracture of the malar with locking of the jaw. Although photographed within three hours of accident the deformity is already masked by swelling and ecchymosis.

They are often sustained during football by the impact of another player's head or as the result of a fall or motor crash. The lines of fracture of an impacted malar correspond fairly closely with the articulations of the bone except that the inferior fracture line passes through the malar process of the maxilla which is included in the displacement (Fig 600). The malar prominence is flattened and the cheek prolapses but the deformity is often concealed by swelling and hæmorrhage. The lateral wall of the antrum is broken; the sinus fills with blood and there is bleeding from the nose. Opacity of the antrum is disclosed in radiographs and by transillumination of the face. The fracture line usually involves the inferior orbital canal and contusion of the infra-orbital nerve causes anaesthesia of the face. The superior alveolar nerves may also be injured. The roof of the antrum is fractured and sinking of the lower and outer part of the orbit causes diplopia. Depression of the zygomatic arch interferes with movement of the coronoid process so that it is difficult to open and close the mouth (Fig 603). The mandible may swing to the opposite side and cause malocclusion of the teeth.

Treatment—The malar should be elevated as soon as possible. The hair of the temporal region is shaved and an incision about 1 in. in length is made within the hair margin anterior and parallel to the superficial temporal vessels. The deep temporal fascia is divided and a long lever such as a periosteum elevator or a Küster's malar lever is passed through the substance of the temporal muscle until it lies beneath the body of the malar. A fulcrum is supplied under the lever by a small rolled bandage and the malar

is raised. The fracture line usually involves the inferior orbital canal and contusion of the infra-orbital nerve causes anaesthesia of the face. The superior alveolar nerves may also be injured. The roof of the antrum is fractured and sinking of the lower and outer part of the orbit causes diplopia. Depression of the zygomatic arch interferes with movement of the coronoid process so that it is difficult to open and close the mouth (Fig 603). The mandible may swing to the opposite side and cause malocclusion of the teeth.



Fig 604

Same case as Fig 603 ten days after replacement of the bone showing the scar within the hair margin and the technique of operation.

¹ R. Mo. 1. Experiences with various Methods of Skeletal Fixation in Fractures of the Jaws. *Proc. Roy. Soc. Med.* 194. xxxv. No. 6.
² Gillies, I. ner and Stone. Fractures of the Malar Zygomatic Compound. *Brit. Jour. Surg.* 1941. xl. 651.

is disimpacted and elevated (Fig 604). The instrument should be passed under all parts of the bone from the zygoma to the orbit and down to the lower margin. It is often unnecessary to use post-operative support, but if the bone tends to redisplace when the lever is removed the antrum should be packed. The gum mucosa is incised behind the canine tooth, and through a small anrostomy the cavity is lightly pricked with 1-in ribbon gauze soaked in pig iodoform co (Whitehead's varnish), the malar bone being held meanwhile in the corrected position. The end of the ribbon gauze is left lying free in the mucous membrane wound. Alternatively reduction may be maintained by passing a wire through the orbital angle of the malar and attaching it to a rod fixed to a plaster head-piece.¹

FRACTURES OF THE PALATAL UNIT

Horizontal separation fracture of the palate—This injury, known as Guern's fracture, closely follows the first line of maxillary weakness described

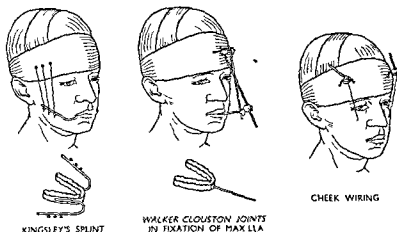


FIG 605

Methods of immobilising the maxilla

by Le Fort (Fig 600). A severe blow centred near the nasal spine separates the palate and drives it backwards. The upper incisors bite behind the lower, and there may also be rotational displacement causing an open bite deformity. The displacement is reduced by gripping the palatal bones on each side at the alveolar margin with special forceps, rocking the bone until it is disimpacted, and replacing it in accurate occlusion with the mandibular teeth. Reduction is maintained by a maxillary cap splint cemented to the teeth and fixed to a Kingsley's splint, or to a maxillary splint with universal joints (Figs 605-607).

Vertical separation fracture of the palate—The clinical diagnosis of midline separation of the palate is obvious because there is a visible split in the mucous membrane of the roof of the mouth, malocclusion of the teeth and a wide gap between the central incisors (Fig 600). First aid treatment consists simply in fixing elastic bands to teeth on opposite sides of the upper

¹ J. W. Gerrie 'Fracture of the Maxillary Zygomatic Compound' *Canal Med Jour* June 1934: 332

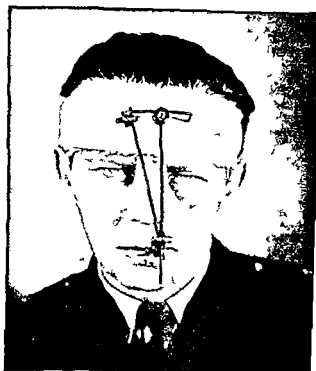


FIG 606

Maxillary splint with Walker Clouston universal joints



FIG 607

Maxillary splint with universal joints showing wire used for traction with Balkan Beam apparatus to bring forward maxilla (it is usually necessary to have a crown piece in plaster head caps—see Fig 603)

jaw This often succeeds in reducing the displacement but if it fails cap splints are fitted to the teeth of the right and left maxillæ with a connecting bar across the palate which can be screwed up and shortened One half of the palate is sometimes displaced upwards as well as outwards and after



FIG 608

Bilateral naso-maxillary fracture in the patient shown in Figs 606-607 The middle third of the face incl. the nasal bones and maxilla is thrust backwards between the malar bone Unless this is held forwards by maxillary splints a disfiguring face deformity remains

reduction by traction the maxilla must be fixed to the mandible by cap splints with lock pins

Palatal-alveolar fracture—One section of the alveolus is separated and the fragment together with the teeth it carries is driven in towards the palate or displaced in the opposite direction (Fig 600) Displacement is reduced by manipulation and fixation is secured by cap splints

END OF VOLUME I